A PROSPECTIVE CROSS-SECTIONAL STUDY OF THE CARDIOVASCULAR CONSEQUENCES IN CHILDREN WITH CHRONIC KIDNEY DISEASE

A dissertation submitted in partial fulfillment of the rules and regulations for the award MD (Branch VII-Pediatrics) degree of The Tamil Nadu Dr. MGR Medical University, Chennai to be held in April 2013

CERTIFICATE

This is to certify that the dissertation entitled **"A prospective cross-sectional study of the cardiovascular consequences in children with Chronic Kidney Disease"** is a bonafide, original work done by **Dr. Dulari Gupta**, during her academic term-**May 2011 to April 2013**, at the Christian Medical College, Vellore, in partial fulfillment of the rules and regulations for the award of MD (Branch VII-Pediatrics) degree of The Tamil Nadu Dr. MGR Medical University, Chennai to be held in April 2013

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AIM

To study the cardiovascular consequences in a prospective cohort of children aged 1-18 years with Chronic Kidney Disease(CKD) with Glomerular Filtration Rate (GFR) < 60 ml/min/1.73 m^2.

CONTENTS

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OBJECTIVES

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Primary Objectives:

1. To study the prevalence of Hypertension both manifest (clinic BP Readings) and masked (Ambulatory BP monitoring).

2. To study prevalence of Left Ventricular Dysfunction in children with CKD using Echocardiogram as a tool.

Secondary Objectives:

1. To study differences in BMI in children on different modalities of treatment for CKD.

2. To compare the cardiovascular risks between children with CKD not requiring dialysis and those on dialysis.

3. To assess the correlation between the calcium-phosphorus product and correlate with risk for vascular calcification.

INTRODUCTION

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Chronic Kidney Disease is an emerging problem worldwide. With increasing awareness about kidney diseases and antenatal ultrasound screening we are detecting more children with congenital anomalies of the kidney and renal tract (CAKUT). In India the prevalence of CKD is estimated to range form 0.79-1.4% in the adult population [1] . The prevalence of CKD amongst Indian children is not known.

The National Kidney Foundation, Kidney Disease Outcome Quality Initiative workgroup has a classification for patients with CKD who are older than two years [2]:

Stage 1 disease — Normal GFR (≥90 mL/min per 1.73 m^2)

Stage 2 disease — GFR between 60 and 89 mL/min per 1.73 m^2

Stage 3 disease — GFR between 30 and 59 mL/min per 1.73 m^2

Stage 4 disease — GFR between 15 and 29 mL/min per 1.73 m^2

Stage 5 disease — GFR of less than 15 mL/min per 1.73 m^2 or end-stage renal disease (ESRD)

As per this definition children with > Stage 3 CKD are categorized to have Chronic Renal Failure (GFR <60 ml/min/1.73m^2). These children develop various morbidities associated with renal failure and need close monitoring of growth parameters. They have special dietary needs and require being on regular medications. Meticulous control of metabolic parameters and avoidance of nephro-toxic drugs can prevent progression to End Stage Renal Disease.

There are many treatment options available for children with chronic kidney disease.Renal replacement in the form of Dialysis or Renal Transplantation is possible and with this supportive care, children with ESRD can survive much longer and can lead near-normal lives. The early age of onset of disease in children with CKD and their increased longevity make them more susceptible to the long term complications of renal failure.

Cardiovascular risks like Hypertension, LV Dysfunction and Atherosclerosis are rare amongst the general pediatric population (< 3%)[3] but in children with CKD are a leading cause of mortality and morbidity [45]. In adolescents with CKD the cardiovascular morbidity is 500-1000 times more than the general population [6].

No studies have been undertaken so far to study the cardiovascular risk factors in Indian children with CKD. More studies and systemic reviews are required in this field to increase our knowledge and to institute preventive strategies to delay progression of renal disease. Identifying children with CKD who are at risk for cardiovascular disease early will help them live longer. Together with better medical management and Renal Replacement Therapy the morbidity and mortality may decrease further. This study was thus designed to determine the cardiovascular risk factors prevalent amongst children with CKD and to identify factors associated with poorer outcomes.

LITERATURE

REVIEW

LITERATURE REVIEW

Chronic Kidney Disease (CKD) is a common problem worldwide and is rapidly increasing due to increasing awareness about kidney diseases and antenatal fetal ultrasound screening. In the USA the CDC has documented CKD in 16.85/1000 of the general population aged > 20 years in the year 1999-2004 which is a 15.9% increase when compared to the years 1988-1994 according to the National Health and Nutrition Examination Survey. In a study conducted by Soylemezoqlu et al in Turkey the prevalence of CKD amongst children aged 5-10 years was found to be 0.94 (95th % CI 0.63-1.35) [7]. According to the US Renal Data System 2011 annual report the incidence of CKD has increased by 1.1% to 355.4 per million populations [8]. In Serbia, amongst 336 children studied Peco Antic et al found the median annual incidence of CKD 2-5 stages was 14.3 per million age-related population (p.m.a.r.p), while those of CKD 2-4 or CKD 5 were 9.1 and 5.7 p.m.a.r.p., respectively [9]. In the Ital Kid study the mean incidence of CKD amongst Italian children was 12.1 per million age related population [10] with a point prevalence of 74.7 per million age related population. In India the prevalence of CKD is estimated to range form 0.79-1.4% in the general population [1] . The prevalence of CKD amongst Indian children is not known.

The treatment of ESRD in Low and Medium Income countries (LMIC) is very difficult due to lack of financial support. In South East Asia, Renal Replacement Therapy costs 10 times the annual per capita income and there is very low coverage of these expenses by health insurance [11]. This high cost of treatment leads to less than 10% of people with ESRD receiving RRT in LMIC.

With the sharp rise in obesity and metabolic syndrome amongst Indian children and adolescents, they too are more susceptible to develop chronic kidney disease [12,13].

Now there are many treatment options available for children with chronic kidney disease. Meticulous control of metabolic parameters and avoidance of nephro-toxic drugs can prevent progression to end stage renal disease. Renal replacement in the form of Dialysis or Renal Transplantation is possible and with this supportive care, children with ESRD can survive much longer and can lead near-normal lives. The early age of onset of disease in children with CKD and their increased life span make them more susceptible to the long term complications of renal failure.

Definition:

Chronic Kidney Disease was defined by the National Kidney Foundation in 2002:

a. Kidney damage for >3 months, as confirmed by kidney biopsy or markers of kidney damage, with or without a decrease in glomerular filtration rate or

b. GFR <60 mL /min/ 1.73 m^2 for >3 months, with or without kidney damage [14].

The National Kidney Foundation, Kidney Disease Outcome Quality Initiative workgroup has a classification for patients with CKD who are older than two years [2]:

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Table 3. Chronic Kidney Disease: A Clinical Action Plan

Stage	Description	GFR (mL/min/1.73 m^2)	Action*
	At increased risk	≥60 (with CKD risk factors)	Screening, CKD risk reduction
1	Kidney damage with normal or ↑ GFR	≥90	Diagnosis and treatment, Treatment of comorbid conditions, Slowing progression, CVD risk reduction
2	Kidney damage with mild ↓ GFR	60–89	Estimating progression
3	Moderate ↓ GFR	30–59	Evaluating and treating complications
4	Severe ↓ GFR	15–29	Preparation for kidney replacement therapy
5	Kidney failure	<15 (or dialysis)	Replacement (if uremia present)

Shaded area identifies patients who have chronic kidney disease; unshaded area designates individuals who are at increased risk for developing chronic kidney disease. Chronic kidney disease is defined as either kidney damage or GFR <60 mL/min/1.73 m^2 for ≥3 months. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies.

* Includes actions from preceding stages.

Abbreviations: GFR, glomerular filtration rate; CKD, chronic kidney disease; CVD, cardiovascular disease

In children < 15 years of age, estimated GFR (eGFR) is calculated using Serum Creatinine values according to the Schwartz Formula. In older children (> 15 years) the Cockcroft-Gault formula is used to calculate eGFR.

Estimated GFR by Schwartz formula is calculated by:

$$\text{GFR mL/min/1.73m}^2 = \frac{k \times \text{Height (cm)}}{\text{Serum Creatinine (mg/dl)}}$$

where k is 0.33 for low-birthweight infants <1 yr old

0.45 for term infants <1 yr old whose weight is appropriate for GA

0.55 for children and adolescent girls

0.70 for adolescent

ETIOLOGY OF CKD:

The causes of CKD amongst children less than 5 years old are most commonly a result of congenital abnormalities such as renal hypoplasia, dysplasia, or obstructive uropathy. Other causes include congenital nephrotic syndrome, prune belly syndrome, cortical necrosis, focal segmental glomerulosclerosis, polycystic kidney disease, renal vein thrombosis, and hemolytic uremic syndrome.

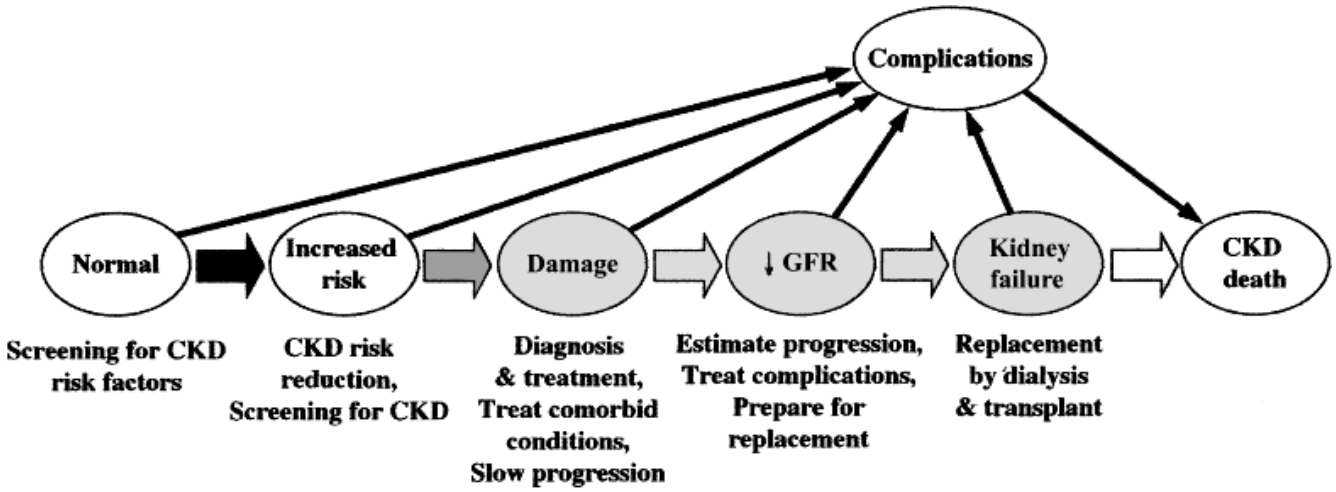
In children older than 5 years of age acquired diseases (glomerulonephritis including lupus nephritis) and inherited disorders (familial juvenile nephronophthisis, Alport syndrome) are more common. Metabolic disorders (cystinosis, hyperoxaluria) and inherited disorders (polycystic kidney disease) can also cause CKD.

The NAPRTCS report showed that congenital causes, including congenital anomalies of the kidney and urinary tract (CAKUT) (48%) and hereditary nephropathies (10%) were the most common causes of CKD amongst children [15].

In a study done by P Hari et al in AIIMS India the etiology of CKD amongst children was found to be [16]:

Cause	Number	Percentage
Chronic Glomerulonephritis	84	27.5
Reflux Nephropathy	51	16.7
Obstructive Uropathy	97	31.8
Neurogenic Bladder	14	4.5
HUS	5	1.6
Renal Dysplasia	15	4.9
Hereditary Nephropathy	20	7.5

Miscellaneous Cortical Necrosis	2	0.6
Unknown	17	5.7



COMPLICATIONS OF CHRONIC KIDNEY DISEASE [17]:

Metabolic acidosis: Kidney is the major organ involved in acid base balance and the main organ that eliminates fixed acids (sulphuric, phosphoric acids) that are produced in the body due to metabolic activity. In renal failure, the inability to eliminate these fixed acids leads to metabolic acidosis. Metabolic acidosis is defined as serum bicarbonate < 20 mEq/L or need for bicarbonate supplementation [18]. The prevalence of metabolic acidosis increases with worsening renal failure. In stage 1 CKD 0.97% children were acidotic, whereas in stage 4 and 5 CKD the prevalence of acidosis was 54.55%.

Growth failure: According to the North American Renal Trial and Collaborative Study 37 % of children with CKD and 47% of children on dialysis had significant short stature [19]. The factors leading to short stature in CKD are non-hormonal factors such as anorexia leading to poor nutrition, electrolyte imbalance, anemia, metabolic acidosis and renal osteodystrophy as well as

hormonal factors such as growth hormone deficiency and resistance [20]. Those with worsening stage of CKD are shorter. A Cochrane review showed that treatment with recombinant human growth hormone resulted in an increase in height velocity amongst children with CKD but the studies were not long enough to predict adult height [21] . In renal transplant recipients, there is some evidence that growth hormone therapy may be detrimental to graft function.

Delayed Puberty: Delayed puberty occurs in children with CKD [18] and partly contributes to the short stature because of the absence of the pubertal growth spurt.

Electrolyte Disturbances: Hyperkalemia can occur due to drugs, metabolic acidosis, and constipation and prolonged fasting. Hyponatremia is more common than hypernatremia. The main cause of hyponatremia is excessive free water intake [22].

Metabolic Bone Disease: Metabolic bone disease is common in advanced stages of CKD in children. It is almost universal amongst children with stage 4 and 5 CKD, while only a minority (6.28%) of children with CKD stage 1 have bone disease. Pathogenesis of metabolic bone disease in CKD is complex. The renal inability to excrete phosphate leads to hyperphosphatemia. This results in lowering of serum calcium levels and stimulates production of parathyroid hormone and this secondary hyperparathyroidism leads to bone resorption. Further there is a decrease in production of 1,25 di-hydroxy vitamin D in CKD because of poor tubular function & hyperphosphatemia. This in turn leads to mineralization defects (osteomalacia). The growing child has a greater need for vitamin D. The markedly reduced 1’25 (OH) D levels in CKD are

particularly harmful to the growing skeleton. Metabolic acidosis per se causes loss of calcium from bone and compounds matters.

Hyperphosphatemia severe enough to warrant use of Phosphate binders, need for active Vitamin D metabolites or PTH levels >70 pg/ml (10 picomoles/L) are markers of bone disease. In renal tubular disease leading to CKD, phosphate wasting from tubular disease [18] is a major cause of bone disease.

Proteinuria: The degree of proteinuria increases with the increasing stage of CKD, and this is independent of the cause of CKD [23].

Anemia: Only 31.25% of Children with CKD stage 1 are anemic while upto 93.3% of those with stage 4 and 5 CKD have anemia. This is an added factor that impedes linear growth in children with CKD.

Hypertension: 18% of children with CKD have overt hypertension as made out with clinic readings whereas an additional 38% have masked hypertension which can only be made out with ambulatory BP monitoring [24]. Sodium and fluid retention, Left Ventricular dysfunction, atherosclerosis and arteriosclerosis all contribute to hypertension.

Fluid Overload: Fluid overload is a common complication in CKD patients specially those undergoing dialysis [25]. Fluid overload, hypertension and anemia contribute to LVH and indirectly to increased cardiovascular mortality.

Atherosclerosis: Normal children rarely ever have atherosclerosis. Early onset atherosclerosis is well studied amongst children with CKD [26].

LVH: Children with CKD have LV Hypertrophy compared to age and sex matched controls. The degree of LV Hypertrophy correlates to the duration of CKD and the stage of CKD [24]

.

Congestive Heart Failure: It is very difficult to differentiate fluid overload state form CCF amongst children on dialysis. Similarly it is equally difficult to differentiate salt and water retention due to inadequate dialysis from signs of CCF. Detection of hypotension during the course of dialysis plays in key role in determining which factor is predominant. Hypotension is a major cause of inadequate ultrafiltration during dialysis.

CARDIOVASCULAR RISKS IN CHILDREN WITH CKD:

Unlike adults, children are less likely to present with angina, myocardial infarction, or ECG abnormalities of Left Ventricular hypertrophy. In view of these differences between adults and children in terms of cardiovascular problems, it is important to evaluate for latent risk factors for cardiovascular disease in children and treat them aggressively to reduce cardiovascular morbidity mortality.

With renal replacement therapy, even though the lifespan of children with CKD has increased considerably, there is a greater susceptibility to cardiovascular morbidity due to the adverse effects of hypertension, fluid overload, left ventricular dysfunction and dsylipidemia.

The traditional risk factors for cardiovascular disease as defined by the Framingham study are:

a. Older age

b. Male sex

c. Hypertension

d. Higher LDL cholesterol

e. Lower HDL cholesterol

f. Diabetes

g. Smoking

h. Physical inactivity

i. Menopause

j. Family history of CVD

k. LVH

Many of the above are not pertinent to the pediatric population.

As outlined by Wong et al **novel non-traditional risk factors/ Uremia related risk factors** for cardiovascular risks amongst children with CKD have also been described (5):

a. Albuminuria

b. Homocysteine

c. Lipoprotein (a) and apolipoprotein(a) isoforms

d. Lipoprotein remnants

e. Anemia

f. Abnormal calcium/phosphate metabolism

g. Extracellular fluid volume overload

h. Electrolyte imbalance

i. Oxidative stress

j. Inflammation (C-reactive protein)

k. Malnutrition

l. Thrombogenic factors

m. Sleep disturbances

n. Altered nitric oxide/endothelin balance

o. Infection (*Chlamydia pneumoniae)*

p. Peripheral Renin-Angiotensin-Aldosterone activity

CARDIOVASCULAR MORTALITY:

Cardiovascular disease is a leading cause of mortality and morbidity amongst children with CKD [45]. Mortality due to cardiovascular causes is rare (less than 3%) amongst the general pediatric population [3]. In adolescents with CKD the cardiovascular morbidity is 500-1000 times more than the general population [6].

In a study done by the European Dialysis and Transplant Association Registry, 41% of deaths in children < 15 years with CKD were due to cardiovascular causes [27]. Identical results were reported by the Dutch retrospective study in which cardiovascular causes accounted for 41% of deaths [28]. The most common causes of cardiovascular mortality were: cerebro-vascular accidents, congestive cardiac failure, myocardial infarction and sudden cardiac death (cardiac arrest) respectively. Data from the Australian and New Zealand Renal Transplant Registry also showed mortality up to 30 times higher than the general population with 45% of the deaths caused by cardiovascular events [29]. However a lower cardiovascular mortality of 23% was

reported by the US Renal Data Systems (a retrospective analysis of the cause of death from 1990-1996 in US children with ESRD) [30].

Study Registry	Author	Number of children Recruited	Mortality due t Cardiovascular Risks (%)	Refere
European Dialysis and Transplant Association Registry	Multiple	5482	41	[31]
Dutch Registry	Groothoff et a	381	41	[32]
Australian and New Zealand Renal Transplant Registry	Mc Donald SF et al	1634	45 (30 times greater	[29]
US Renal Data Systems	Parekh et al	1380	23	[33]

According to Parekh et al in the US Renal Data System the most common cardiac causes of mortality amongst adults was coronary artery disease followed by congestive cardiac failure, whereas amongst children sudden cardiac death (cardiac arrest) arrhythmias and cardiomyopathy respectively are the commonest causes of mortality [33].

PATHOPHYSIOLOGY OF CARDIOVASCULAR DISEASE [3]:

Cardiac remodeling occurs which leads to left ventricular (LV) Hypertrophy. There are two main types of LVH:

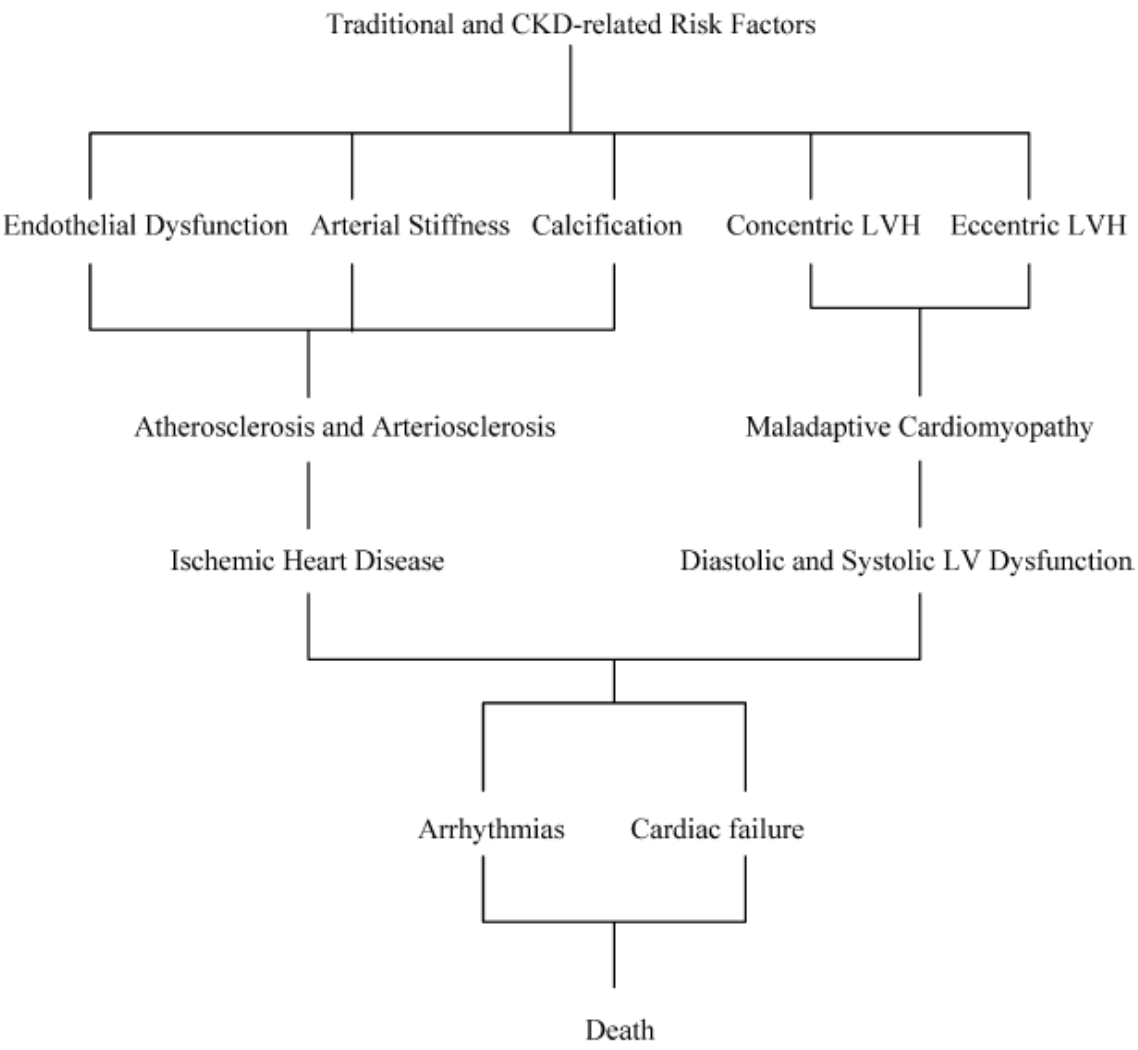
1. **Concentric LVH** occurs in condition of pressure over-load secondary to long standing hypertension.

2. **Eccentric LVH** occurs in condition of volume overload due to sodium retention, anemia, A-V Shunts [34].

Pressure hypertrophy causes a parallel pattern of sarcomere arrangement. This results in increased wall thickness and concentric LVH.

Volume overload causes sarcomeres to be arranged in series with an increase in the longitudinal cell growth. The LV dilatation is disproportionate to the wall thickness leading to eccentric LVH.

The flow chart given below summarizes the changes in the heart in CKD.



Atherosclerosis is the other major contributor to cardiovascular disease. It involves the formation of an atheromatous plaque in the intima of the artery. The plaque is formed by fat containing macrophages called foam cells. Patchy irregular dystrophic calcification of the plaque ensues once the plaque ruptures. This calcification is due to several factors such as metabolic acidosis, inflammation and mechanical shear stress, the Ca X P product which tends to be high due to elevated serum phosphate in CKD, and treatment of renal osteodystrophy with 1,25 dihydroxy Vitamin D and calcium citrate. There are other mechanisms involved in the process of

calcification. For example, the levels of serum Fetuin A, an effective inhibitor of calcium-phosphorus complex formation is lowered in children with CKD.

Measurements of the carotid intima-medial thickness or inducible myocardial ischemia detected by coronary stress test are reliable markers for the presence of atherosclerosis.

The endothelial progenitor cells help in the repair of damaged intima and prevent plaque formation. However in children with CKD there is a reduction in the number of Endothelial Progenitor cells and this impairs the process of endothelial repair and thus contributes to atherosclerotic damage.

Arteriosclerosis is increased arterial rigidity of the entire arterial tree. It involves both intimal and medial involvement. It increases the wall thickness and the length of the arteries in turn leading to an increase in systolic BP, arterial thickening and pulse pressure.

HYPERTENSION:

Hypertension:

Up to 18 % of children with CKD have confirmed hypertension, whereas an additional 38% can have masked hypertension [35]. The cause of hypertension is multi-factorial: fluid overload, Left Ventricular dysfunction, atherosclerosis and arteriosclerosis [4]. Hypertension is a very important risk factor in progression of CVD in CKD children since it is amenable to early detection and treatment.

White Coat HT:

White Coat HT is more pronounced amongst children than in adults. Children are more likely to have high BP readings in a Clinic setting, with normal BP values outside the clinic. To detect true HT amongst children, clinic BP readings are not adequate. This can lead to over diagnosis of HT and undue treatment with Anti HT medications.

Masked HT:

Masked hypertension is defined as Clinic BP less than 95th centile for age and sex, but Mean SBP by ABPM greater than 95th centile and SBP Load greater than 25 %. Masked HT cannot be detected by clinic BP readings alone and requires ABPM for detection.

Ambulatory BP Monitoring (ABPM):

ABPM helps to circumvent the problem of White coat HT. It allows BP monitoring while the child is in his/her own surroundings and doing their normal day to day activities. Most importantly it measures night time BP levels which are important predictors of cardiovascular outcomes.

It is considered the gold standard in detecting hypertension amongst CKD patients. Ambulatory BP Monitoring has been found to be more sensitive in detecting latent Hypertension than Clinic BP readings [36].

In the ESCAPE trial ABPM was found to co-relate closely with Home BP Monitoring when compared to Clinic BP readings [37]. One fourth of the children with HT can be missed when only Home BP Monitoring is used. Thus ABPM is the best means of identifying all CKD children

with hypertension. This method also avoids a mistaken diagnosis of hypertension in those with only white coat HT.

In India, Sinha et al have shown the usefulness of ABPM amongst children with CKD [38].

Suggested Schema for APBM in Children [39]:

Classification	Clinic BP*	Mean Ambulatory SBP†	SBP Load, %[70,75]
Normal BP	<95th percentile	<95th percentile	<25
WCH	>95th percentile	<95th percentile	<25
Masked hypertension	<95th percentile	>95th percentile	>25
Prehypertension	>95th percentile	<95th percentile	25–50
Ambulatory hypertension	>95th percentile	>95th percentile	25–50
Severe ambulatory hypertension (at risk for end-organ damage)	>95th percentile	>95th percentile	>50

Modified from Lurbe et al,[74] with permission.
BP indicates blood pressure; SBP, *systolic* blood pressure.
*Based on the National High Blood Pressure Education Program Task Force Standards.
†Based on ABPM values of Soergel et al or the smoothed values of Wühl.

End Organ Damage:

ABPM is a predictor for target organ damage and can predict future outcomes.

Left Ventricular Hypertrophy co-relates best with SBP and BP Index detected by ABPM [39]. ABPM picks up early loss of nocturnal dip in BP and it is a better predictor of end organ damage [37]. Loss of normal nocturnal dipping is associated with greater proteinuria and poorer renal function [40,41]. ABPM was found to be a better predictor of proteinuria than home BP, single clinic BP by automated device, or standard BP monitoring [42].

ABPM Indices Measured:

Average BP readings: Mean, Daytime, Night time

BP Load: the percentage of valid ambulatory BP measures above the 95th percentile of BP for age, gender, and height. BP Load of 25-30% is considered significant.

BP Index: The average BP for each patient divided by the 95th centile BP value specific for that patient.

Nocturnal Dipping: >= 10% fall in SBP and DBP from daytime to night time. It can be calculated by ([mean daytime ABPM−mean nighttime ABPM] / (mean day ABPM) ×100)

Normative ABPM Data:

There is a separate normative data for pediatric ABPM which is different from the usual Clinic BP guideline in children [37,43–46] . The largest study was done by O Sullivan et al amongst healthy school children in Newcastle [45]. The data is arranged according to gender, height and age. We have used the data by Wuhl et al as recommended by the American Heart Association [37] to interpret our ABPM readings because normative data for Indian children are not available.

Country	**Author**	**Number of children**	**Age Groups (Years)**	**Reference**
Germany: Escape Trial Group	Wuhl et al	118	3-19	[37]
USA	Harshfield GA et al	300	10-18	[43]
Spain	Lurbe et al	241	6-16	[44]
UK Newcastle	O Sullivan et al	1121		[45]
Germany	Reichert H et al	564	9-13	[46]

LEFT VENTRICULAR DYSFUNCTION:

Children with CKD are known to have cardiac dysfunction [47]. Yet there are few deaths amongst children with CKD due to cardiovascular diseases. Thus we need other intermediate end points to determine cardiovascular risks amongst children. Left Ventricular Hypertrophy is a predictor of mortality amongst adults with CKD [48]. Left Ventricular Hypertrophy helps to improve cardiac contractility and lower wall stress in view of increased after-load and preload [49].

Many studies in adults have shown that CKD leads to systolic dysfunction and progressive heart failure [50]. In children however systolic LV Dysfunction rarely occurs [5152].

Mark Mitsnefes et al conducted a study of LV Dysfunction amongst 25 children with CKD and found that LV Mass Index, LV Performance and contractility at rest was increased in children with CKD. The children on dialysis had lower contractile reserve during exercise which might be an early indicator for severe systolic dysfunction. They also demonstrated that LV dysfunction developed early in CKD and progressively worsens with decreasing levels of GFR [49].

Amongst children on dialysis worse LV dysfunction was seen in children on hemodialysis with respect to children on peritoneal dialysis [53].

LVM Definitions:

LVM is calculated according to the formula by Devereux [54]:

LVM (g) = 0.8 {1.04 [($LVEDD$ + PWT +$IVST$)3 -($LVEDD$)3] + 0.6}

LVEDD: Left Ventricular End Diastolic Diameter

PWT: Posterior Wall Thickness

IVST: Inter Ventricular Septal Thickness

LVH was earlier defined as LVM > 38.6 gm/m2.7 based on the study by National High Blood Pressure Education Program Working Group on High Blood Pressure in Children

and Adolescents [55]. But this did not take into account variations due to the height, age or gender of the child. Khoury et al defined LVH as LVMI greater than the 95th centile for gender and chronological age [56]. Whereas Foster et al took LVH as LVMI greater than 95th cenitle for height age (age of the child with the same height growing at 50th height percentile) [57]. Here we have followed the last definition since it most accurately represents the relation of height to LVM.

Country	**Authors**	**Number of children**	**Results**	**Referenc**
USA New York Cincinnati	De Simone et al 1992	444 (4-23 mo)	LVM/Ht^2.7 had least least correlation wit Ht	[58]
USA Cincinnati	Daniels et al 1995	192 (6-17 yrs)	LVM/Ht ^2.7 best correlated with LVM/LBM	[59]
USA Boston Cincinnati	Foster et al 2008	440 (0-21 yrs)	LVMI inversely correlated with Ht, LVM centile curves superior to LVMI for normalizing LVM in children	[57]
USA Cincinnati	Khoury et al 2009	2273 (0-18 yrs)	LVMI for age centile curves superior to LVMI in normalizing LVM in children	[56]

Left Ventricular Hypertrophy is defined as LV mass greater than 95th centile for age, sex and height [60].

Left Ventricular Mass Index was calculated to account for LVH and body size. It was calculated by dividing the LV Mass by the height raised to a power of 2.7.

The relative wall thickness was measured to determine if LVH was concentric or eccentric. Concentric LVH was defined as increased LVM Index with RWT > 0.41, whereas eccentric LVH was defined as increased LVM Index with normal RWT < 0.41 [61]. Concentric hypertrophy results in stiffer LV and impaired diastolic filling. Eccentric hypertrophy is more likely due to volume overload.

All children with CKD have diastolic dysfunction, which is worse in children on dialysis. This diastolic dysfunction can lead on to LV Dysfunction and later to CCF.

ATHEROSCLEROSIS:

Atherosclerosis is one of the known classical risk factors for cardiovascular disease [62]. It is commonly seen in children with CKD, but the stage of CKD when atherosclerosis begins is not exactly known. Intima/ media thickness of large arteries such as the carotid artery determined by Doppler studies is an early predictor of atherosclerosis. Normal children almost never have atherosclerosis. Thus any amount of atherosclerosis detected can be attributed to be secondary to CKD. The Carotid intimal thickness can be measured by non-invasive methods like USG Doppler or by CT of the neck [63]. UG Doppler is preferred since there is no risk of radiation exposure.

Carotid Intimal Medial thickness(c IMT) is measured in the internal carotid artery at a fixed point 20 mm distal to the carotid bifurcation. It is compared to age and sex matched normograms [64].

In a study done by Jun Oh et al amongst 39 adults with childhood onset of CKD, they found that coronary calcification was present in 92% of patients and carotid intimal thickness was increased when compared to age and sex matched controls. The **carotid intimal thickness** strongly correlates with the **Calcium X Phosphorus product** and the cumulative dialysis duration [65]. It is also related to the prevalence of Chlamydia pneumoniae, increased CRP, PTH levels and levels of plasma homocystine [66]. The calcium-phosphorus product also correlates with the vascular remodeling and non-compliance of vessels [67].

In a study done in pediatric CKD patients who died, as many as 60% were found to have evidence of soft tissue calcification[68]. The degree of calcification was directly correlated with Vitamin D supplementation. Children who received Calcitriol had increased calcification when compared to those who received Dihydrotachysterol or D2, D3 supplementation only.

The risk of early onset atherosclerosis with mortality due to cardiovascular causes is 10-30 times greater in children on dialysis than in children without CKD [67]. Children on hemodialysis had greater elevation of carotid IMT than those on peritoneal dialysis [69]. Post renal transplantation children had greater carotid IMT when compared to those on dialysis [70].

A summary of a review of studies showing a significant increase in c IMT amongst children with CKD compared to controls [26] is presented below:

DYSLIPIDEMIA:

Elevated Lipid profile is a known classical risk factor for cardiovascular disease. Children with CKD have been found to have abnormally elevated lipid profiles as compared to age and sex matched controls [65]. In a study by Rafat M Muhaisen et al in Gaza amongst 112 children studied,

children with CKD showed significantly higher levels of cholesterol (163.6±39.8 vs. 141.8±24.2 mg/dL, *P*<0.0001), triglycerides (145.5±67.1 vs. 82.9±39.8 mg/dL, *P*<0.0001), low-density lipoprotein (92.6±31.9 vs. 72.5±19 mg/dL, P<</i>0.0001). These children were also found to have lower levels of high-density lipoprotein (41.9±11.0 vs. 52.7±11.7 mg/dL, *P*<0.0001) compared to controls [71].

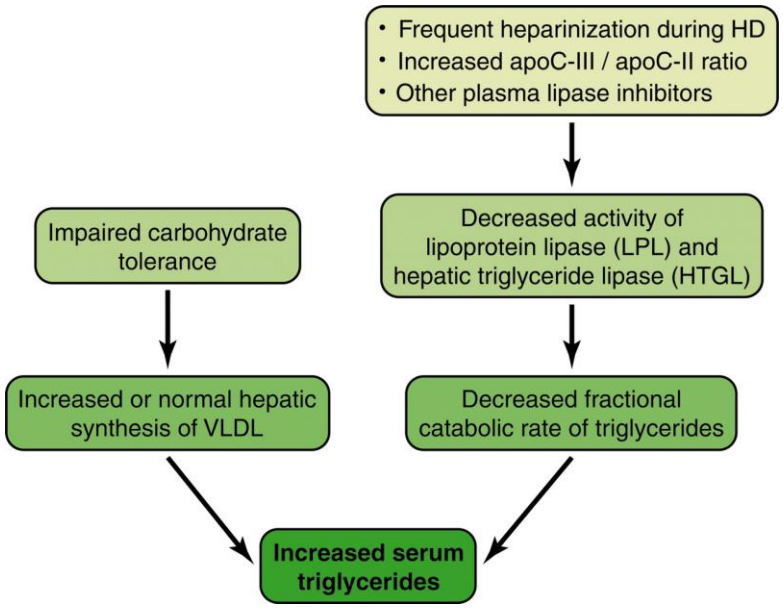
It is evident that patients on hemodialysis are better off so far as several lipid parameters are concerned

The usefulness of statin therapy has been proven amongst adults with early CKD but not amongst those requiring dialysis. The AURORA [72] and the 4D trial (Die Deutsche Diabetes Dialysis Study) [73]

Have shown that statins have the required lipid lowering effect, but they do not significantly reduce mortality due to cardiovascular causes amongst adults on dialysis [74].

Thus dyslipidemia is another modifiable risk factor for cardiovascular disease in children with CKD which needs to be studied in greater detail.

Pathophysiology of hypertriglyceridemia in uremia [75]:



The present study will help in outlining the cardiovascular risks amongst children with CKD form a teaching hospital in India. The present study was undertaken because no studies have been done so far in India. There is no Cochrane Review available on the risks of cardiovascular disease amongst children with CKD. Thus more studies and systematic reviews are required in this field. This will increase our knowledge base about the cardiovascular risks in children with CKD, help identify children with CKD who are at risk for cardiovascular disease and thus help in improving our management of these risk factors. Children with CKD are living longer with better medical management and Renal Replacement Therapy. In order to prevent cardiovascular morbidity and mortality, we need to be aware that CKD children have increased cardiovascular risks and we need to manage these risk factors better

METHODOLOGY

METHODOLOGY

This was a prospective cross-sectional study conducted in the Pediatric Department of Christian Medical College and Hospital Vellore from Oct 2011- Nov 2012. CMCH is a tertiary care center located in South India which is a referral center for local patients as well as patients from all over India.

In our hospital 6000 children come to the Pediatric Nephrology OPD each year. Out of these 50 new cases of CKD were seen from March 2010- September 2011. Thus there are a large number of children with CKD who can be included in our study.

Study Subjects:

Children, aged 1-18 years with Chronic Kidney Disease who presented to the Pediatric Nephrology OPD, General Pediatric OPD or Inpatient Facilities were recruited.

Chronic Kidney Disease as defined by the National Kidney Foundation in 2002: (1) kidney damage for >3 months, as confirmed by kidney biopsy or markers of kidney damage, with or without a decrease in glomerular filtration rate (GFR), or (2) GFR <60 mL /min/ 1.73 m^2 for >3 months, with or without kidney damage [14].

The Glomerular Filtration Rate was calculated according to the Schwartz Formula for children < 15 years old. For older children the Cockcroft-Gault formula would be used to estimate GFR.

The children were divided into the following groups according to their Glomerular Filtration Rates:

Stage 3 disease — GFR between 30 and 59 mL/min per 1.73 m^2

Stage 4 disease — GFR between 15 and 29 mL/min per 1.73 m^2

Stage 5 disease — GFR of less than 15 mL/min per 1.73 m^2 or end-stage renal disease (ESRD)

Children with GFR < 15 ml/min/1.73 m^2 on Dialysis

Inclusion Criteria:

All children with CKD and GFR < 60 ml/min/1.73 m^2 aged 1-18 years will be recruited after parental consent

Exclusion Criteria:

Any child with Renal, other solid organ, Bone marrow Transplantation

Cancer/ HIV diagnosed during the past 12 months

Genetic Syndromes

Presence of any congenital/structural heart disease or myocardial disease

Methodology:

Informed consent was taken from all parents by the Principal Investigator (Annexure I). A detailed history including demographic data, duration and course of disease, treatment details was taken. A complete physical examination was done including anthropometric data, edema, Clinic Blood Pressure recordings. Information was entered into a Clinical Research Form (Annexure II).

Routine Blood tests (Hemoglobin, Sodium, Potassium, Creatinine, Urea, Calcium, Phosphorus, Alkaline Phosphatase, Bicarbonate, Parathyroid hormone levels) were noted using the P 800 Roche Hitachi Modular System. The mean of 3 clinic BP readings over the previous 3 visits were recorded. Ambulatory BP recording was performed for each child over a 24 hour period using the Spacelabs Healthcare (040-1546-00) machine. ECHO was done to determine Left Ventricular Dysfunction (LV mass, LV Mass Index, Relative Wall Thickness). The Philips IE 33 model with 12 size pediatric probe was used.

The study was approved by the Institutional Review Board of CMC Hospital Vellore.

Ambulatory BP Monitoring:

ABPM was monitored for every child using the Spacelabs Healthcare (040-1546-00) machine. The appropriate sized BP cuff was attached to the non-dominant arm of the child. BP readings were recorded for a 24 hour period. The exact sleep and wake timings of children were not determined, and it was assumed that they sleep between 10 pm to 6 am. Subjects were asked to continue all their normal daily activities. BP recordings were measured every 20 minutes during the day time and every hourly at night time.

Ambulatory HT was defined as BP greater than the 95th centile for age and height [39] as provided in Annexure V. BP load was calculated as the percentage of BP readings above the 95th centile. Nocturnal dipping was defined as a > 10% difference between day time and night time BP readings. Ambulatory BP Index was calculated as the average BP of the child divided by the 95th centile Ambulatory BP for that particular age, height and gender.

ECHO:

ECHO was done for every child using the Philips IE 33 model with 12 size pediatric probes. The ECHO was performed by a trained technician or doctor according to their protocol. The parameters recorded were LVID, LV Posterior Wall thickness, Inter Ventricular Septal Thickness, Relative Wall Thickness, Fractional Shortening, Ejection Fraction, Maximum E velocity, Maximum A velocity and the E/A ratio.

LV Hypertrophy was defined as the LV Mass greater than the 95th centile for age, sex and Height as shown in Annexure IV.

These were used to calculate the LV Mass according to the Devereox formula. The LV Mass Index was calculated as well as the LVM/Ht^2.7.

Sample Size Estimation:

N, Number in each arm of study = $\frac{2 \times SD^2 (Za+Zb)^2}{(m1-m2)^2}$

SD= Standard Deviation

Za= 1.96

Zb= 0.84

M1=Mean 1

M2= Mean 2

LVMI of children with CKD not requiring Dialysis 37.7 +/- 9.9

LVMI of children with CKD with Dialysis 45.7 +/- 10.7

N = $\frac{2 \times 10 \times 10 \times (2.8)^2}{(8)^2}$ = 24.5

Thus 25 children need to be recruited in each arm of the study. Since we have two arms in the study the total sample size was estimated to be 50 children.

Data Analysis:

The data collected was analyzed using SPSS software and chi square tests of significance were done.

RESULTS

RESULTS

Forty Six children with Chronic Kidney Disease were recruited from the Pediatric Nephrology Unit of Christian Medical College Vellore during the study period Nov 2011 to Nov 2012.

The cardiovascular risk factors were analyzed in all these children.

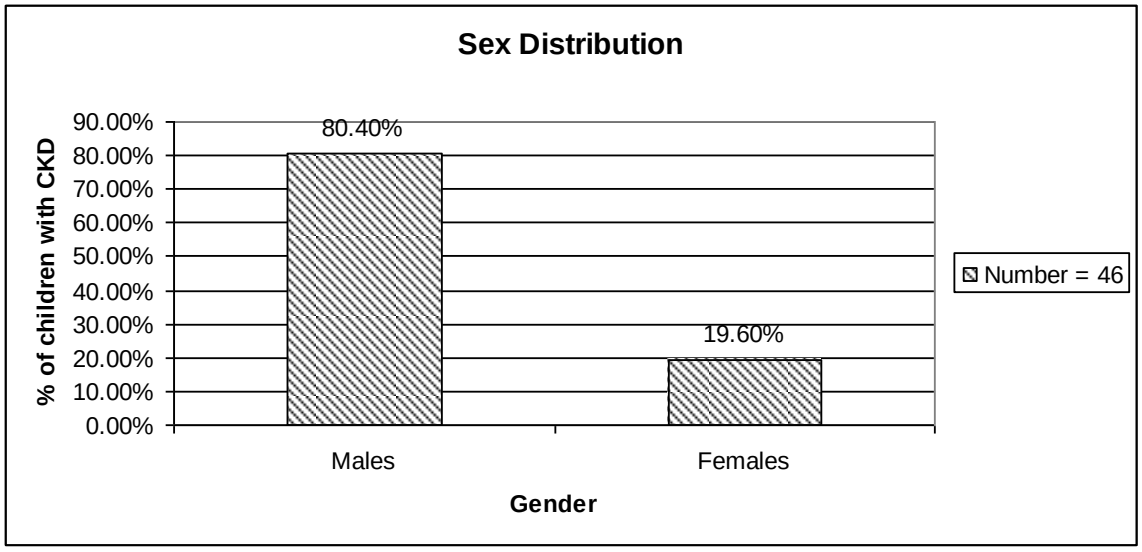
Details of the results obtained are given below:

SEX DISTRIBUTION

Table 1

Sex	Number	Percentage
Boys	37	80.4
Girls	9	19.6
Total	46	100

Figure 1



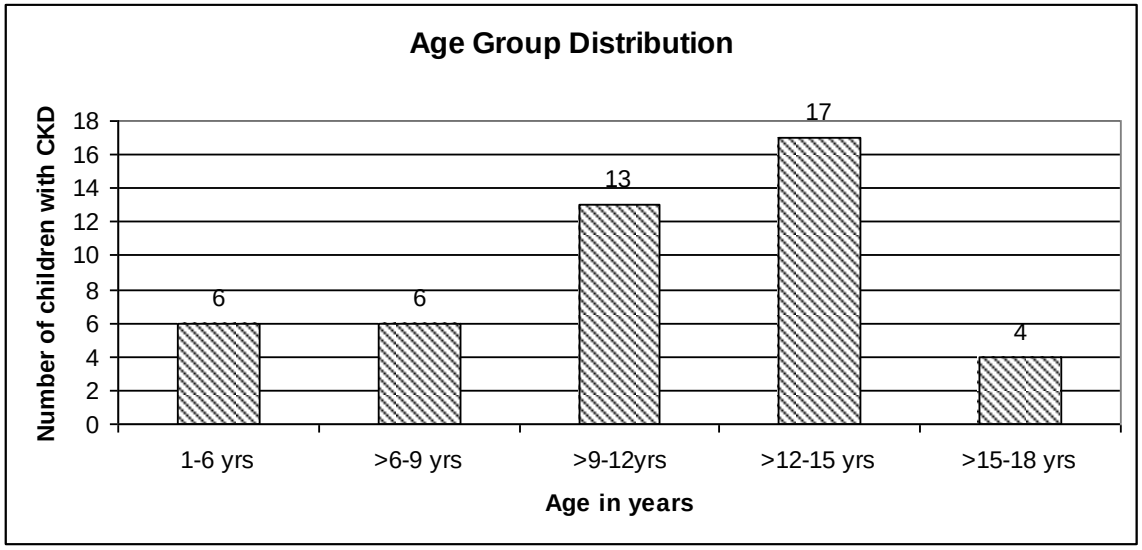
Majority 37/46 (80.4%) of the children with CKD were males.
Male: Female Ratio was 4.1:1

AGE DISTRIBUTION

Table 2

Age Group	Number	Percentage
1-6 yrs	6	13.0
>6yrs to 9yrs	6	13.0
>9 yrs to 12 yrs	13	28.3
> 12 yrs to 15 yrs	17	36.9
>15 yrs to 18 yrs	4	8.6

Figure 2



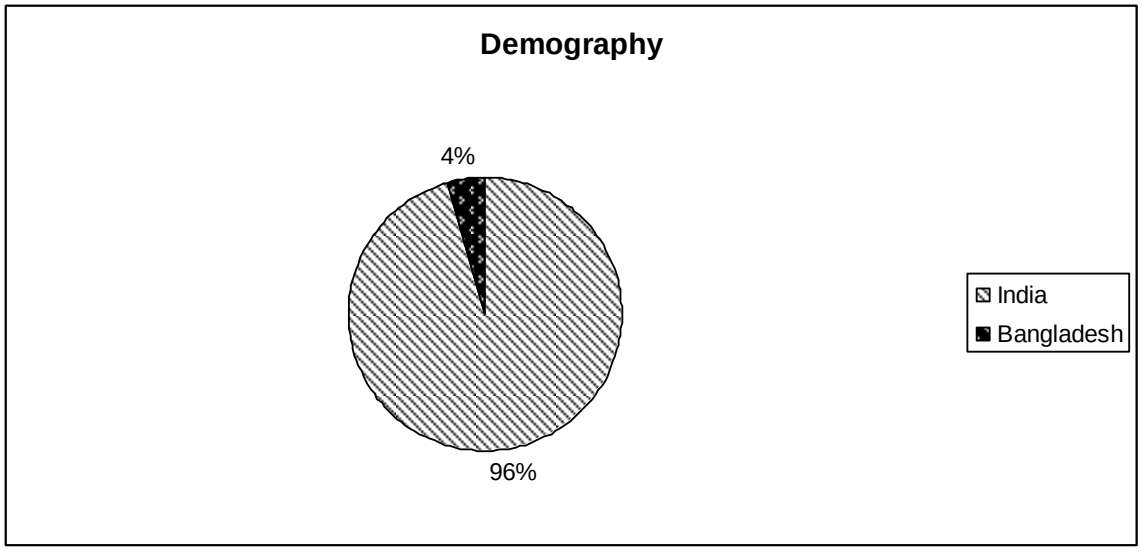
Majority of children presented above 9 years of age
36.9% (17/46) of the children were in the age group of 12-15 years
28.3% (13/46) were 9-12 years old
13% (6/46) were 1-6 years old and 6-9 years old respectively
8.6% (4/46) children were > 15 years of age

DEMOGRAPHY

Table 3

Country	Number	Percentage
India	44	95.6
Bangladesh	2	4.3
Total	46	100

Figure 3



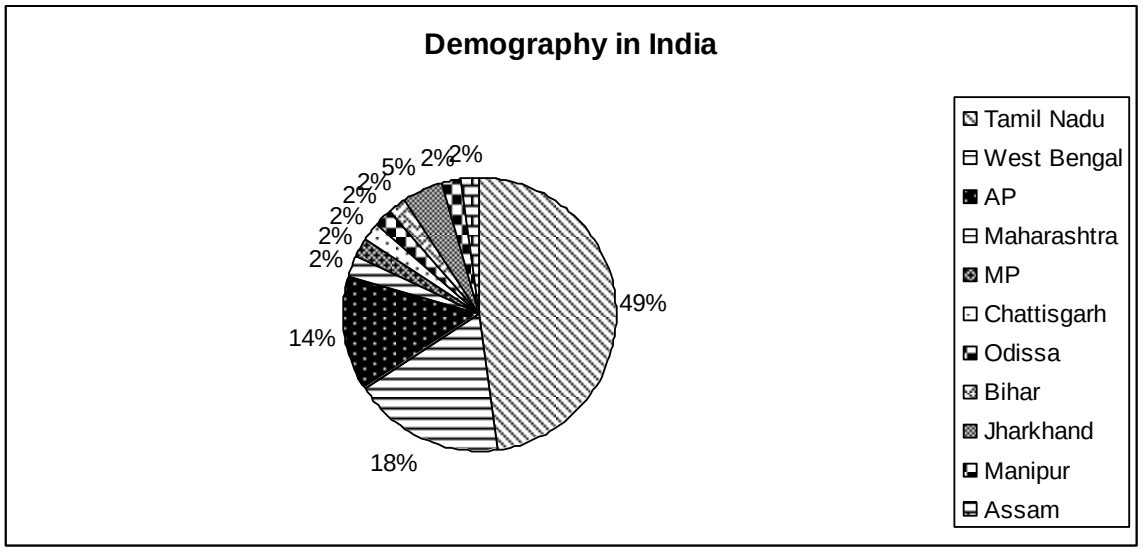
Of the 46 children with CKD studied, 44/46 (96%) were from India.
2/46 (4%) of the children were from Bangladesh.

DEMOGRAPHY FROM INDIA

Table 4

State	Number	Percentage
Tamil Nadu	21	47.7
West Bengal	8	18.2
Andhra Pradesh	6	13.6
Jharkhand	2	4.5
Maharashtra	1	2.3
Madhya Pradesh	1	2.3
Odissa	1	2.3
Chattisgarh	1	2.3
Bihar	1	2.3
Assam	1	2.3
Manipur	1	2.3
Total	44	100

Figure 4



Of the 44 children from India, 21/44(47.7%) were from Tamil Nadu.

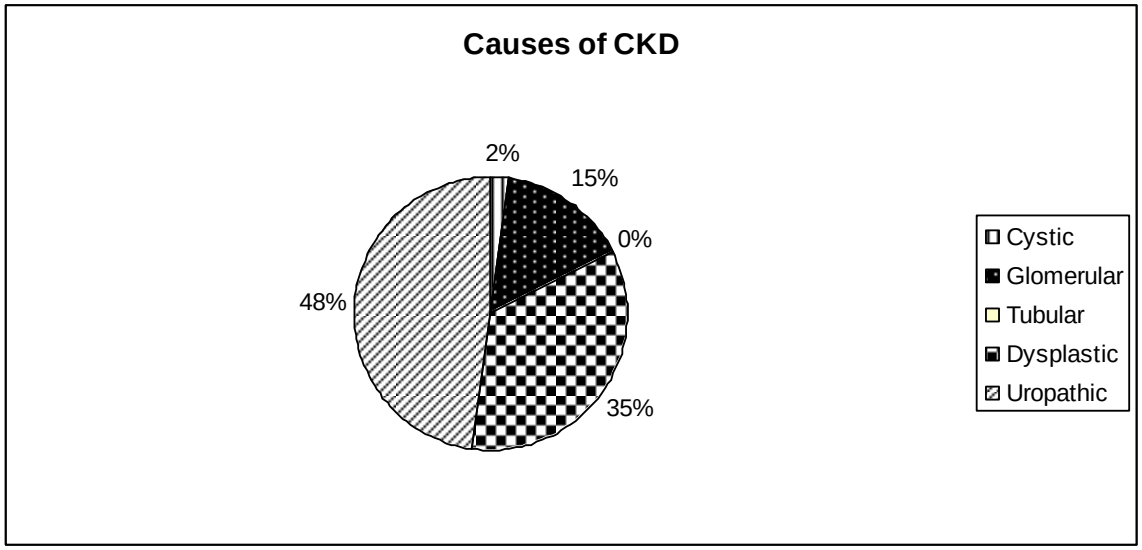
Amongst the children from other states, 8/44 (18.2%) were from West Bengal, 6/44 (13.6%) were from Andhra Pradesh, 2/44 (4.5%) were from Jharkhand and 1 (2.3%) child each from Maharashtra, Madhya Pradesh, Odissa, Chattisgarh, Bihar, Assam and Manipur.

CAUSES OF CHRONIC KIDNEY DISEASE

Table 5

Cause	Number	Percentage
Cystic	1	2
Glomerular	7	15
Tubular	0	0
Dysplastic	16	35
Uropathic	22	48
Total	46	100

Figure 5



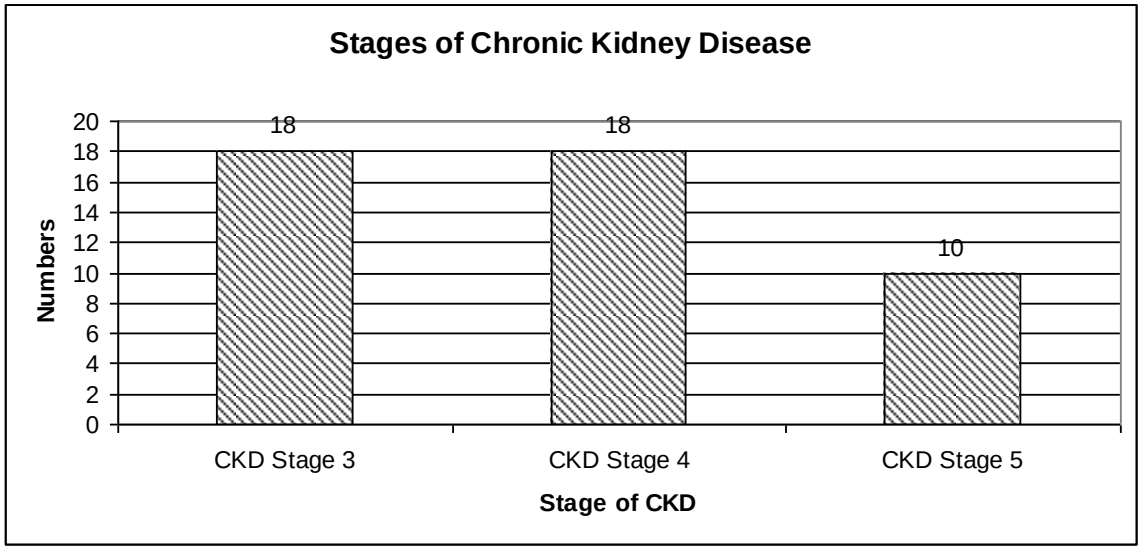
Obstructive Uropathy was the commonest cause of CKD amongst children 22/46 (48%)
Dysplastic kidneys were the second most common cause of CKD 16/46 (35%)
Glomerular causes were found in 7/46 (15%) of children with CKD
Cystic causes were found in 1/46 (2%) of children with CKD

STAGES OF CHRONIC KIDNEY DISEASE

Table 6

Stage of CKD	Number	Percentage
CKD Stage 3	18	39.1
CKD Stage 4	18	39.1
CKD Stage 5 and Dialysis	10	21.7
Total	46	100

Figure 6



Of the 46 children with CKD, 18/46 (39.1%) were in CKD Stage 3 and 4 respectively, while 10/46 (21.7%) had CKD Stage 5 or were on Dialysis.

Out of all the 46 children studied, 42 (91.3%) were found to have cardiovascular risk factors. Details are given below:

Cardiovascular Risks	Percentage in children with CKD (%)
Low BMI	30/42 (71.4%)
Anemia	22/42 (52.4%)
Proteinuria	18/42 (42.9%)
Abnormal Ca X PO4 Product	20/42 (47.6%)

Further analysis was carried out to analyze the cardiovascular risks amongst children with CKD. The children were divided into two groups for the purpose of analysis:

1. Children with CKD not requiring Dialysis
2. Children with CKD on Dialysis

The complications of CKD were compared between the two groups.

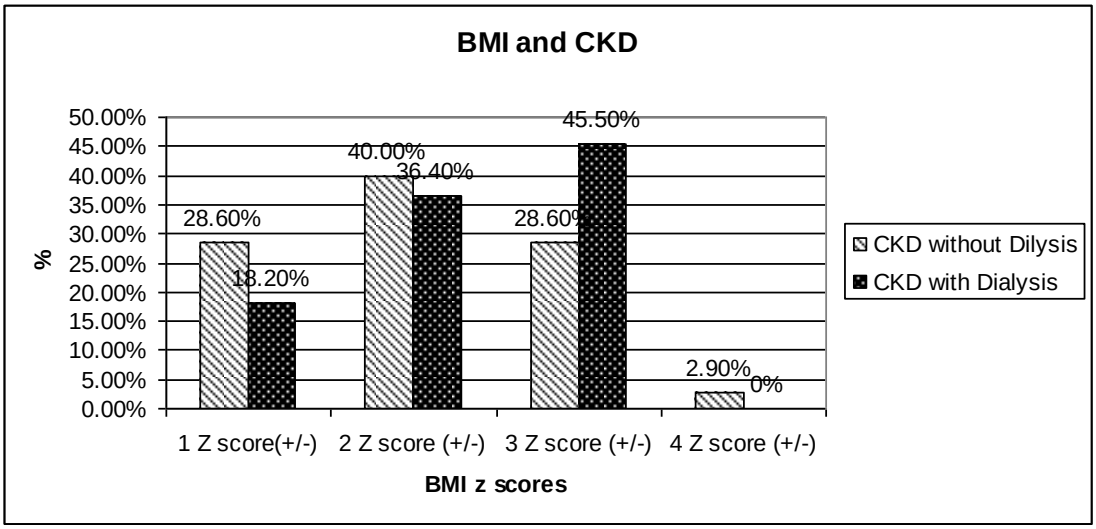
Body Mass Index

Body Mass Index was plotted for each child according to the WHO BMI charts.
A z score from -1 to +1 was considered normal. Further z scores were all abnormal.
Lower z scores correlated with under-nutrition, while higher z scores signified obesity.

Table 7

BMI	CKD without Dialysis	CKD with Dialysis	Total
-1 to +1 Z score	10 (28.6%)	2 (18.2%)	12
-2 to +2 Z score	14 (40%)	4 (36.4%)	18
-3 to + 3 Z score	10 (28.6%)	5 (45.5%)	15
-4 to + 4 Z score	1 (2.9%)	0	1
Total	35	11	46

Figure 7



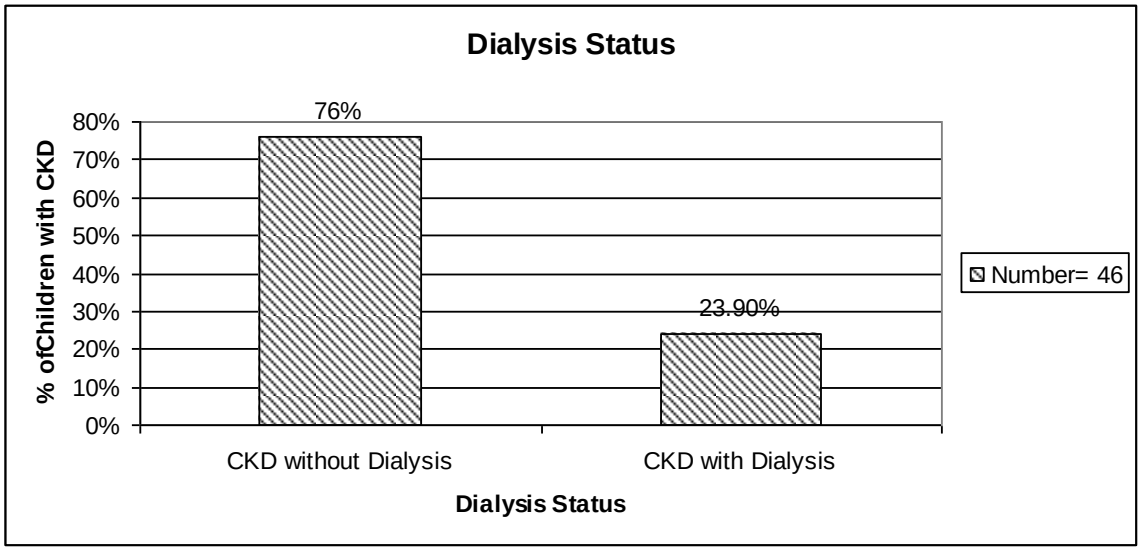
All children except one had normal to low BMI. Only one child had obesity with BMI of 27.3.
Though children in both groups had low BMI, those on dialysis had a lower BMI (81.9%) compared to children in the non-dialysis group (71.5%).

Children with CKD on Dialysis and those not requiring Dialysis

Table 8

Dialysis Status	Number	Percentage
CKD not requiring Dialysis	35	76
CKD with Dialysis	11	23.9
Total	46	100

Figure 8



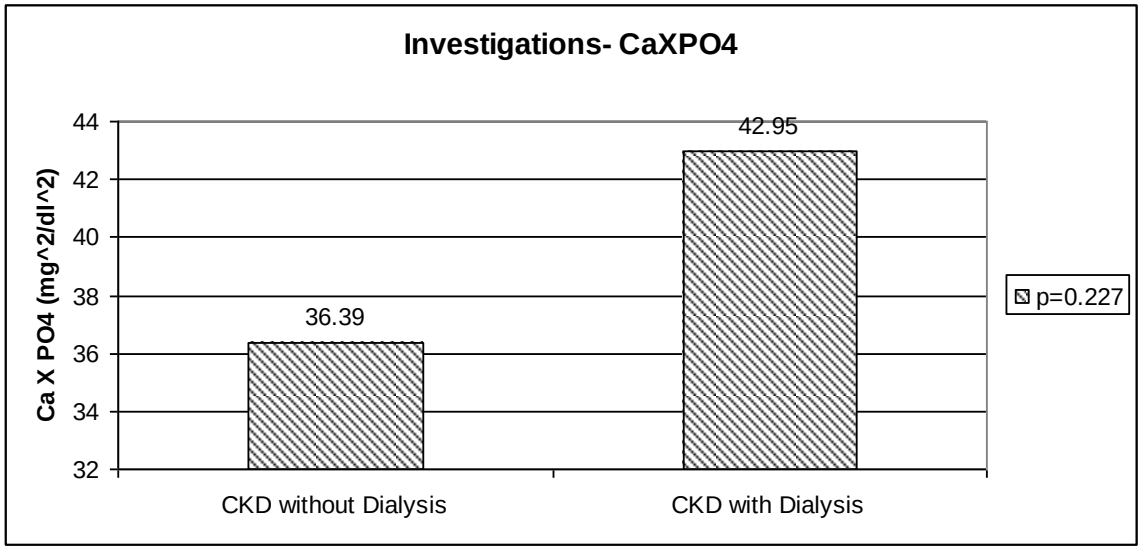
35/46 (76%) of children with CKD were not on Dialysis.
11/46 (23.9%) of children with CKD were on Dialysis.

Calcium Phosphorus Product and CKD

Table 9

	CKD without Dialysis	CKD with Dialysis	P Value
Ca X PO4 Product	36.39	42.95	0.227

Figure 9



There was no significant difference between the Calcium X Phosphorus Product between children with CKD without Dialysis (Ca X PO4 36.39) and children requiring Dialysis (Ca X PO4 42.95 p=0.227).

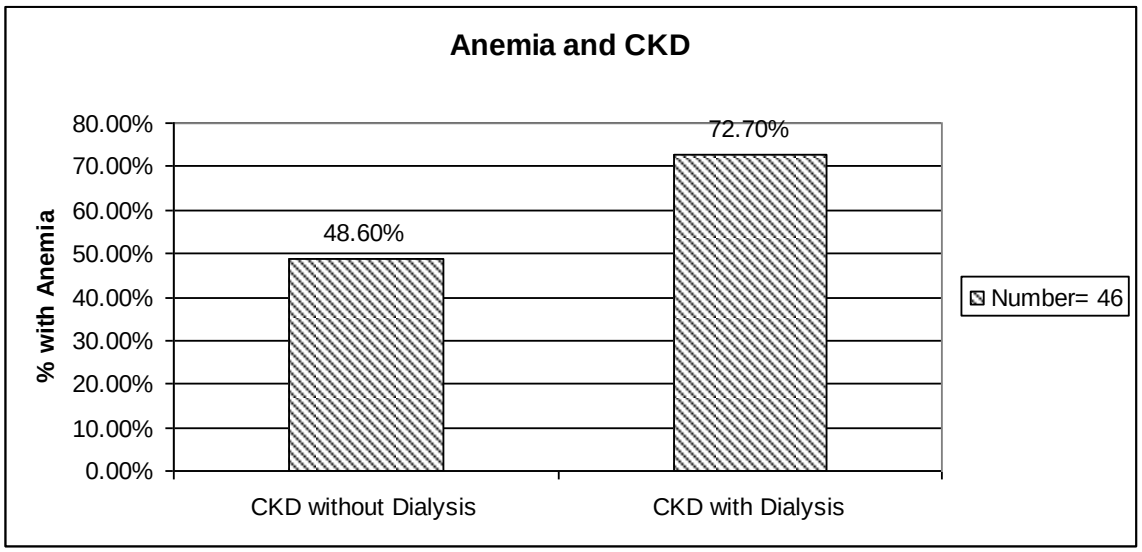
Anemia and CKD

Anemia is defined as hemoglobin levels less than 11 gm%.

Table 10

	Anemia Present	Anemia Absent	Total
CKD without Dialysis	17/35 (48.6%)	18/35 (51.4%)	35
CKD with Dialysis	8/11 (72.7%)	3/11 (27.3%)	11
Total	25	21	46

Figure 10



There was no significant difference in the prevalence of anemia amongst children with CKD without Dialysis 17/35 (48.6%) and children on dialysis 8/11 (72.7%) (p=0.145).

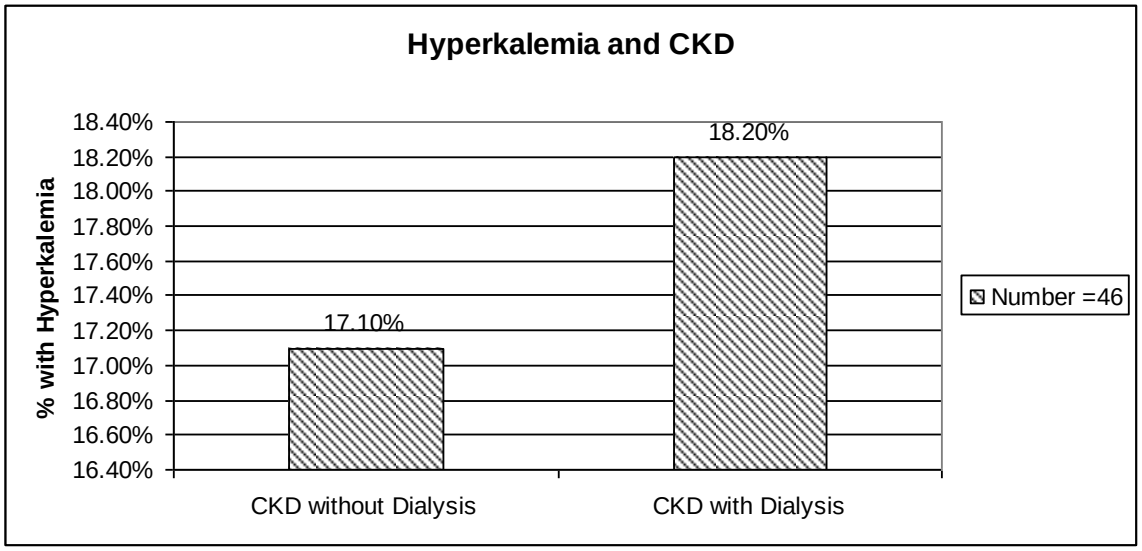
Hyperkalemia and CKD

Hyperkalemia was defined as Potassium levels greater than 5.5 mg/dl.

Table 11

	Hyperkalemia Present	Hyperkalemia Absent	Total
CKD without Dialysis	6/35 (17.1%)	29/35 (82.9%)	35
CKD with Dialysis	2/11 (18.2%)	9/11 (81.8%)	11
Total	8	38	46

Figure 11



There was significant difference in the prevalence of Hyperkalemia amongst children with CKD not requiring dialysis 6/35 (17.1%) and children on dialysis 2/11 (18.2%) **(p=0.026).**

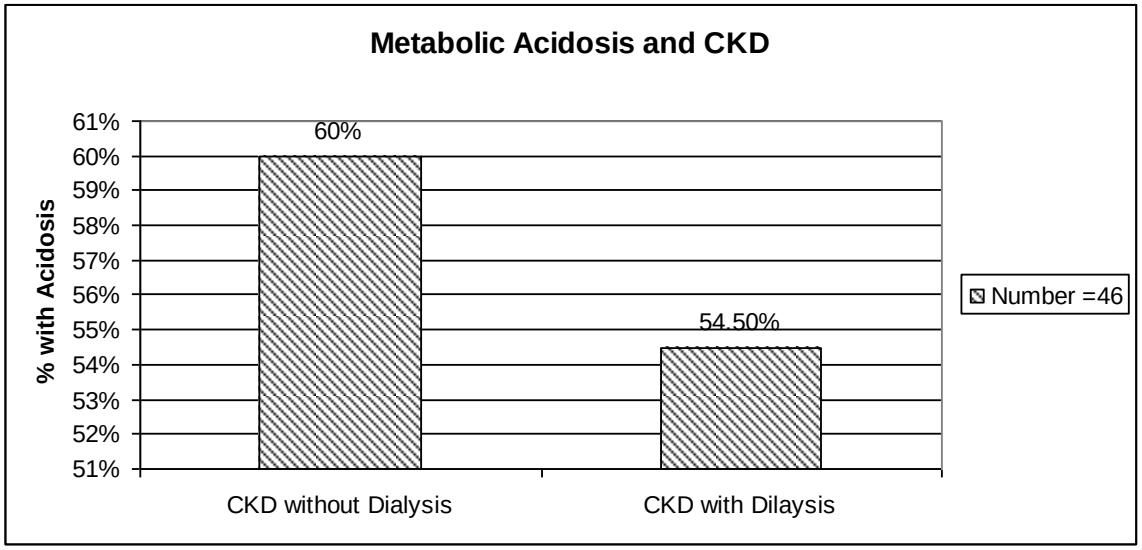
Metabolic Acidosis and CKD

Metabolic acidosis was defined as a serum bicarbonate level lower than 20 mg/dl.

Table 12

	Acidosis Present	Acidosis Absent	Total
CKD without Dialysis	21/35 (60%)	14/35 (40%)	35
CKD with Dialysis	6/11 (54.5%)	5/11 (45.4%)	11
Total	27	19	46

Figure 12



There was no significant difference in the prevalence of Metabolic Acidosis between children with CKD not requiring dialysis 21/35 (60%) and children on dialysis 6/11 (54.4%) (p=0.508).

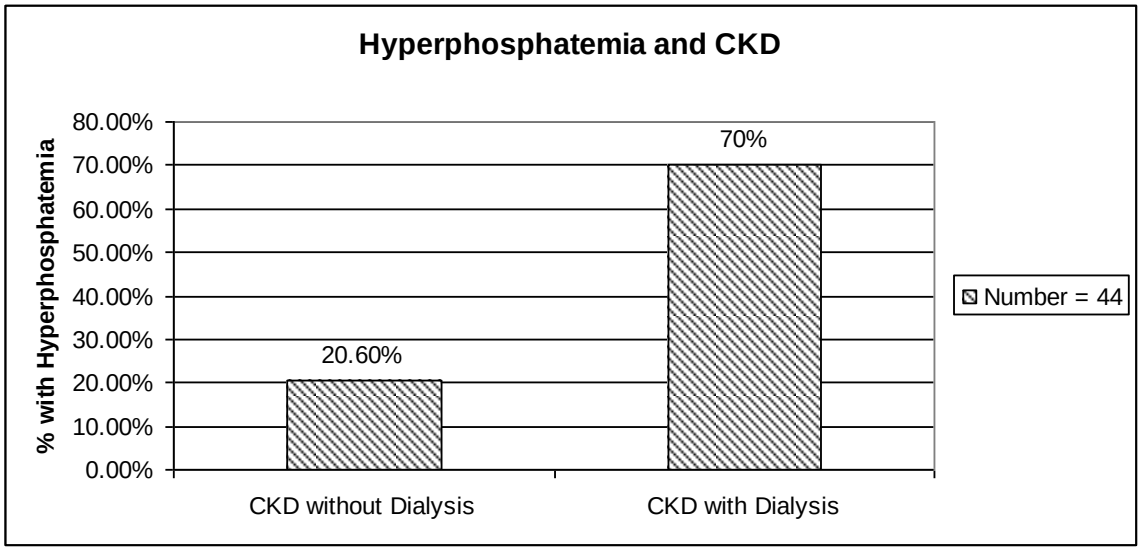
Hyperphosphatemia and CKD

Hyperphosphatemia was defined as serum Phosphorus levels greater than 5 mg/dl.

Table 13

	Hyperphosphatemia Present	Hyperphosphatemia Absent	Total
CKD without Dialysis	7/34 (20.6%)	27/34 (79.4%)	34
CKD with Dialysis	7/10 (70%)	3/10 (30%)	10
Total	14	30	44

Figure 13



There was a significant difference in the prevalence of Hyperphosphatemia amongst children with CKD not requiring dialysis 7/34 (20.6%) and children on dialysis 7/10 (70%) **(p=0.006).**

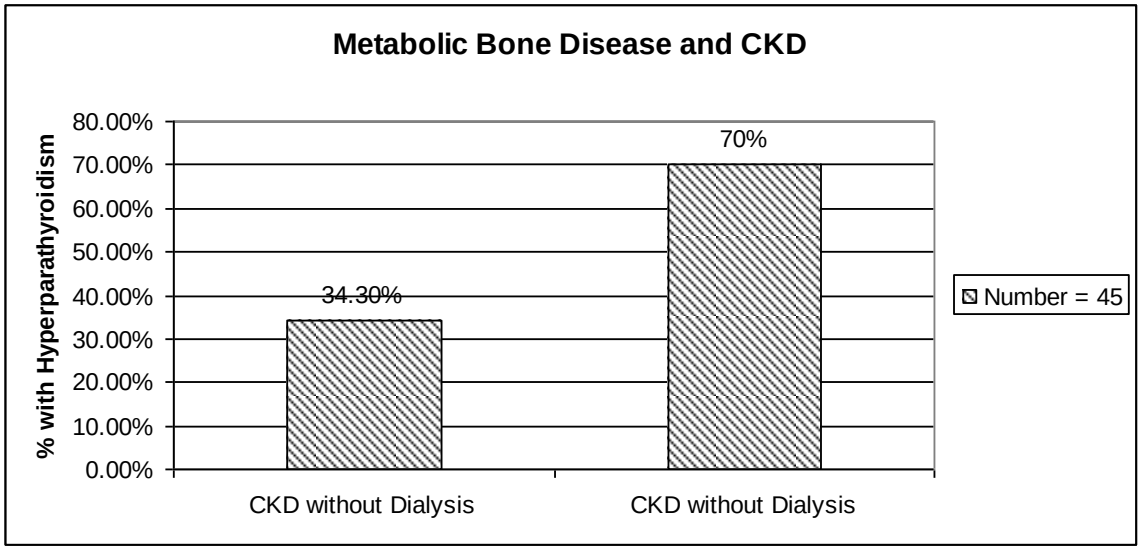
Metabolic Bone Disease and CKD

Metabolic Bone Disease was considered in all children with CKD who had Hyper-parathyroidism with serum PTH levels greater than 250 mg/dl.

Table 14

	Hyper parathyroidism Present	Hyper parathyroidism Absent	Total
CKD without Dialysis	12/35 (34.3%)	23/35 (65.7%)	35
CKD with Dialysis	7/10 (70%)	3/10 (30%)	10
Total	19	26	45

Figure 14



Children on dialysis had a greater prevalence of Metabolic Bone Disease 7/10 (70%) as compared to children with CKD not requiring dialysis12/35 (34.3%) **(p=0.05).**

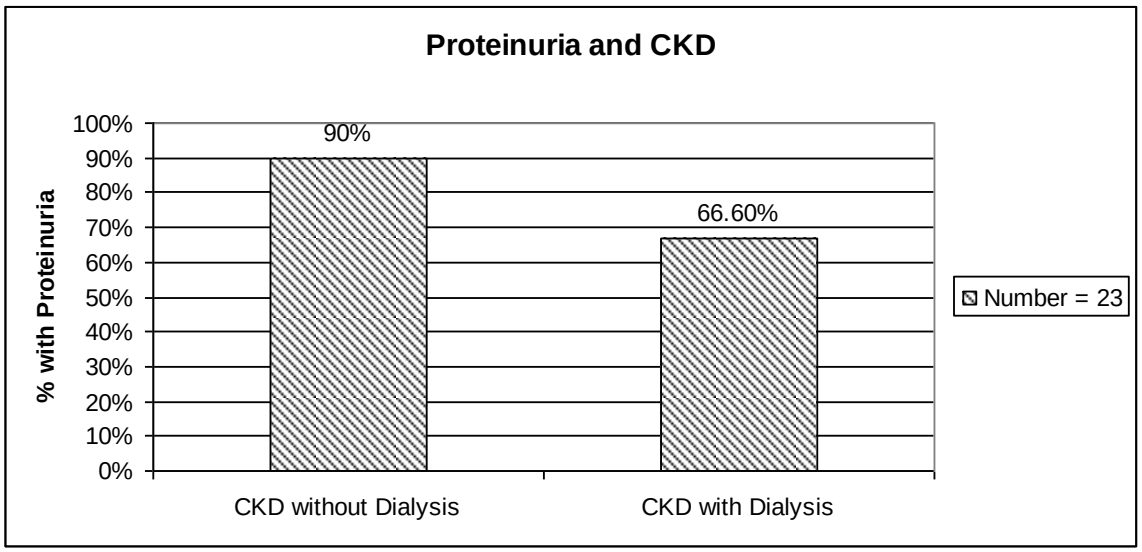
Proteinuria and CKD

Proteinuria was defined as a Urine protein/Creatinine ratio of greater than 0.03.

Table 15

	Proteinuria Present	Proteinuria Absent	Total
CKD without Dialysis	18/20 (90%)	2/10 (10%)	20
CKD with Dialysis	2/3 (66.6%)	1/3 (33.3%)	3
Total	20	3	23

Figure 15



There was no significant difference in the prevalence of Proteinuria between children with CKD not requiring dialysis 18/20 (90%) and children on dialysis 2/3 (66.6%) (p=0.356).

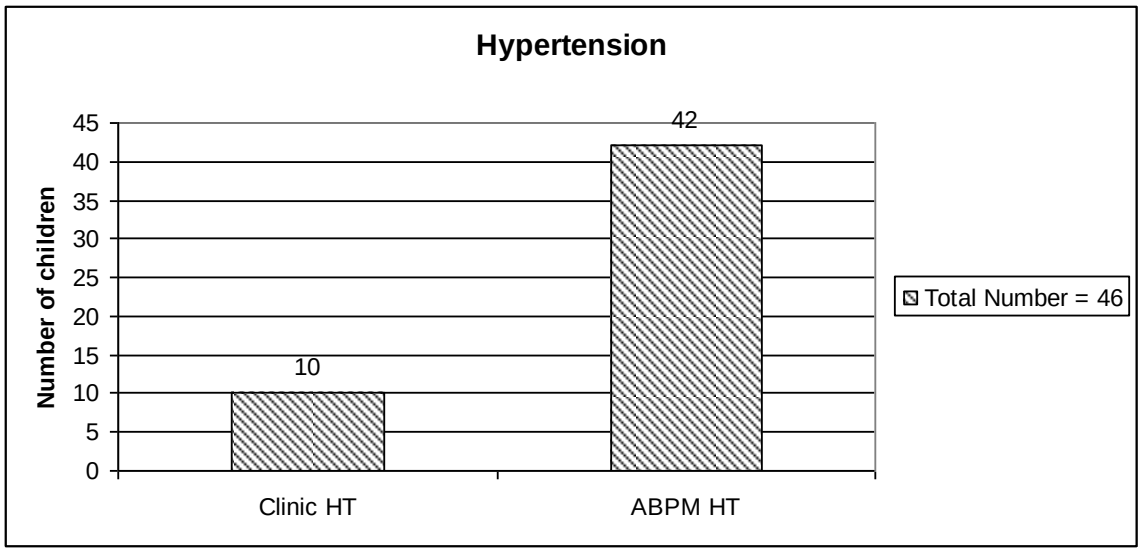
Hypertension and CKD

Hypertension was assessed by Clinic BP readings and by Ambulatory BP Monitoring over a 24 hour period in all children.

Table 16

Hypertension	Present	Absent	Total
Clinic HT	10/46 (21.7%)	36/46 (78.3%)	46
ABPM	42/46 (91.3%)	4/46 (8.7%)	46

Figure 16



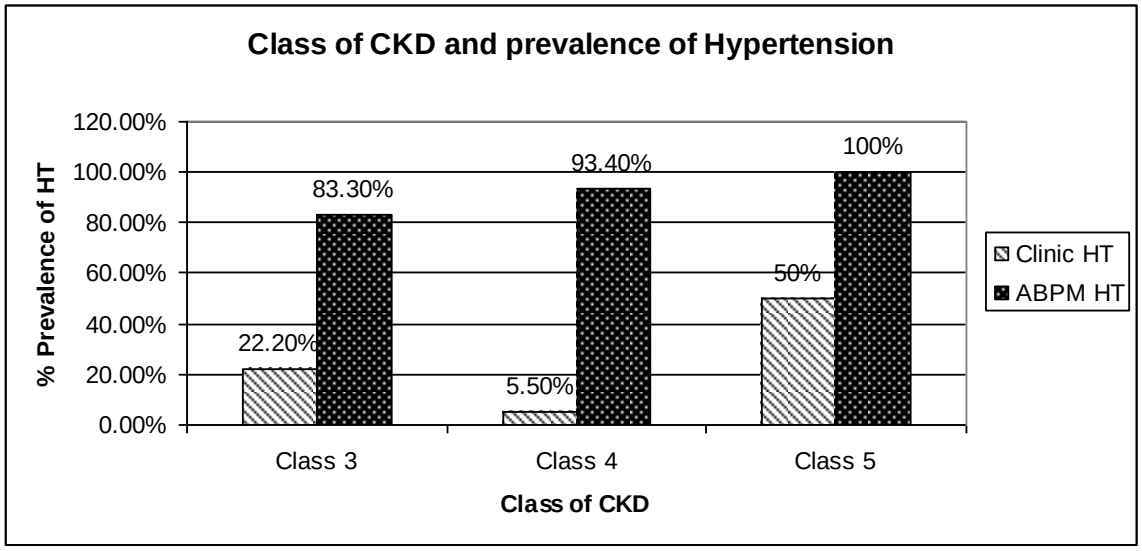
Using Clinic BP alone 10/46 (21.7%) of children with CKD were found to be hypertensive.
With the additional usage of ABPM 42/46 (91.3%) of children with CKD were found to be hypertensive, an additional 32/46 (69.5%) were found to have masked hypertension.

Class of CKD and Hypertension

Table 17

Class of CKD	Clinic HT	ABPM HT	Total
Class 3 CKD	4/18 (22.2%)	15/18 (83.3%)	18
Class 4 CKD	1/18 (5.5%)	17/18 (94.4%)	18
Class 5 CKD and Dialysis	5/10 (50%)	10/10 (100%)	10
Total	10	42	46

Figure 17



ABPM was able to detect more hypertension across all classes of CKD wrt clinic BP readings.

Clinic BP and Number on Anti-Hypertensive Medications

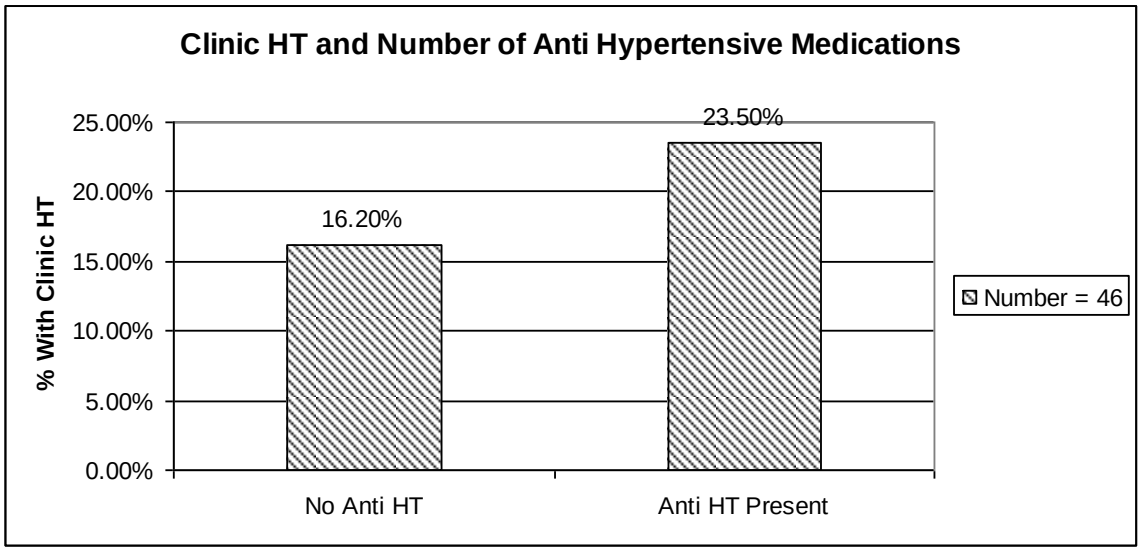
The prevalence of Clinic HT was compared with the number of anti hypertensive medications that the children were taking.

The adequacy of BP control with anti-hypertensive medications was assessed.

Table 18

	No Anti HT	Anti HT Present	Total
Clinic BP Normal	10/12 (83.3%)	26/34 (76.4%)	36
Clinic HT	2/12 (16.6%)	8/34 (23.5%)	10
Total	12	34	46

Figure 18



Of those with Clinic Hypertension, 2/12 (16.6%) were not on any anti hypertensive medications, while 8/34 (23.5%) were on anti hypertensive medications.

2/10 (20%) of children were newly detected to have Clinic HT.

8/10 (80%) of the children with Clinic HT were on anti hypertensive medications and their BP was not adequately controlled.

Ambulatory Hypertension and Anti Hypertensive medication Usage

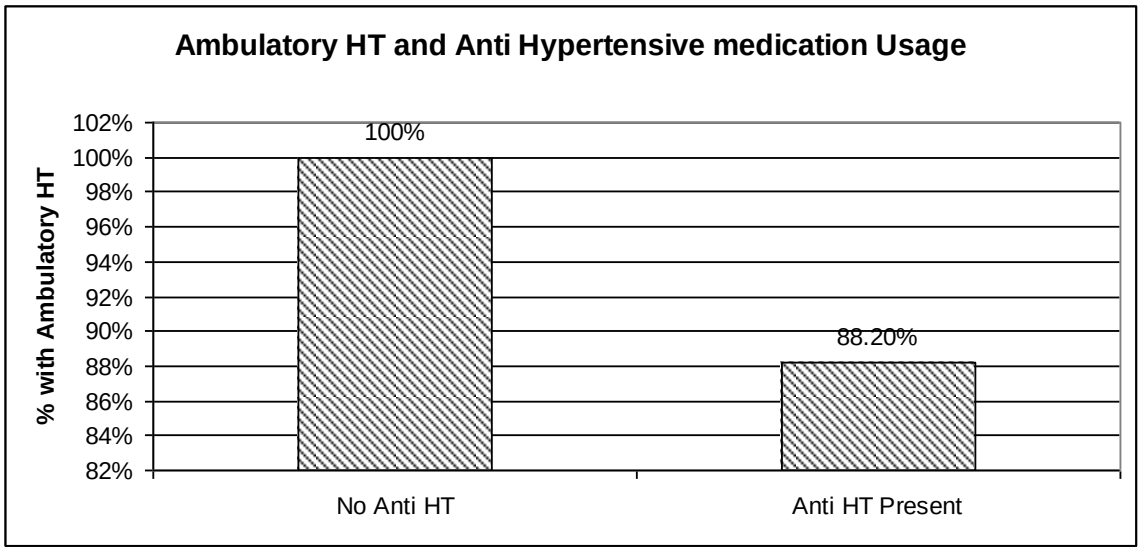
The prevalence of Ambulatory HT was compared with the number of anti hypertensive medications that the children were taking.

The adequacy of BP control with anti-hypertensive medications was assessed.

Table 19

	No Anti HT Medications	**Anti HT Medications Present**	**Total**
Ambulatory HT Absent	0	4/34 (11.8%)	4
Ambulatory HT Present	12/12 (100%)	30/34 (88.2%)	42
Total	12	34	46

Figure 19



There was no significant difference between the prevalence of Ambulatory HT amongst those on anti HT medications 30/34 (88.2%) or those not on any anti HT medications 12/12 (100%) (p= 0.284).

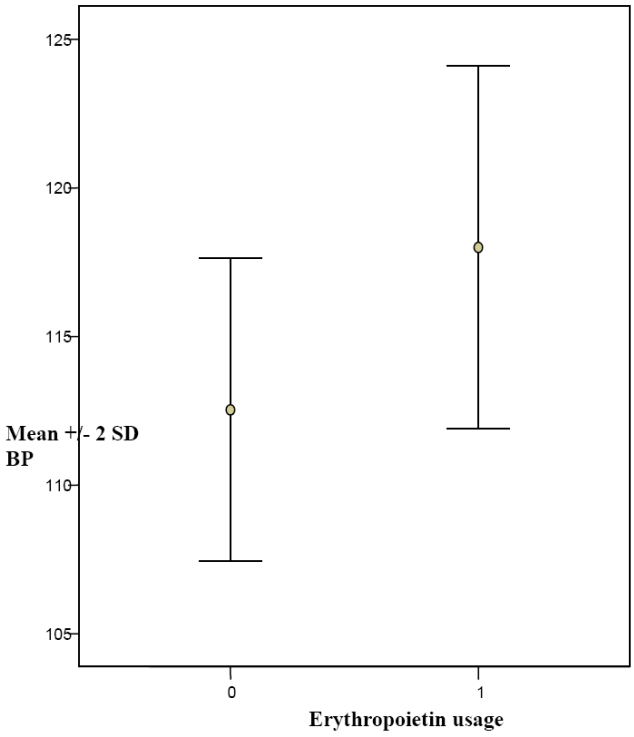
Four children in whom Ambulatory HT was absent were on anti hypertensive medications and their HT was adequately controlled.

Erythropoietin usage vs Hypertension

Table 20

	Erythropoietin	Number	Mean	Std. Deviation
SBP	Absent	32	112.53	14.418
	Present	14	118.00	11.429
DBP	Absent	32	70.00	11.433
	Present	14	76.21	9.316
MBP	Absent	32	84.44	11.086
	Present	14	90.14	9.330

Figure 20



Children on treatment with Erythropoietin were found to have higher SBP, DBP and MBP compared to those not receiving Erythropoietin.

Cardiovascular Dysfunction

ECHO was done in all children to determine LV Dysfunction and to calculate the LV Mass.

LV Hypertrophy was defined as LV Mass greater than 95th centile for age and sex.

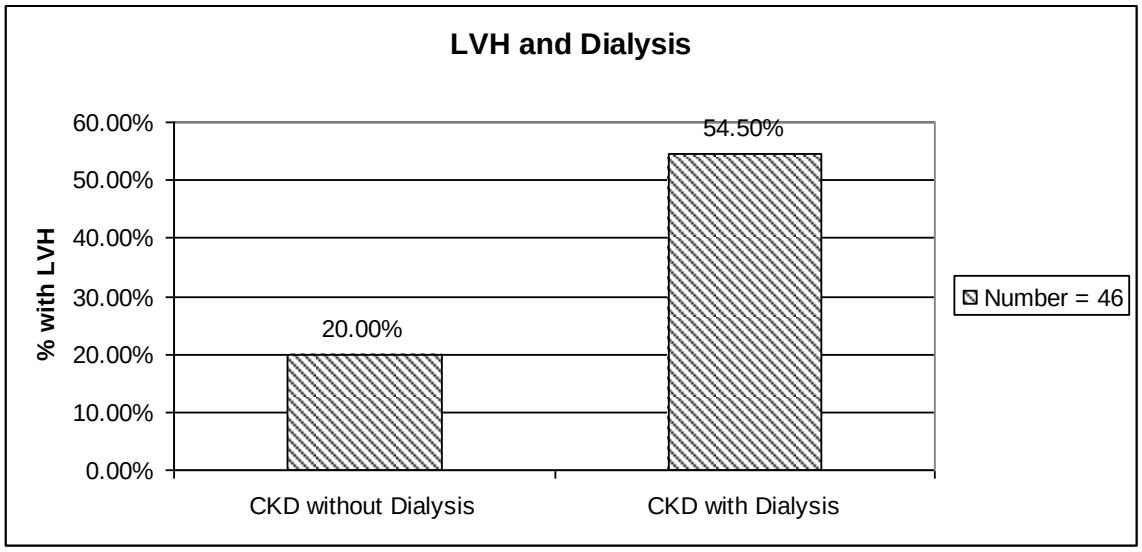
Here we have compared the prevalence of LVH as per the Class of CKD.

LVH and CKD

Table 21

	CKD without Dialysis	CKD with Dialysis	Total
LVH Absent	28/35 (80%)	5/11 (45.4%)	33
LVH Present	7/35 (20%)	6/11 (54.5%)	13
Total	35	11	46

Figure 21



There was a significant difference in the prevalence of LVH amongst children on dialysis 6/11 (54.4%) and children with CKD not requiring dialysis 7/35 (20%) **(p=0.036).**

LVH v s ABPM

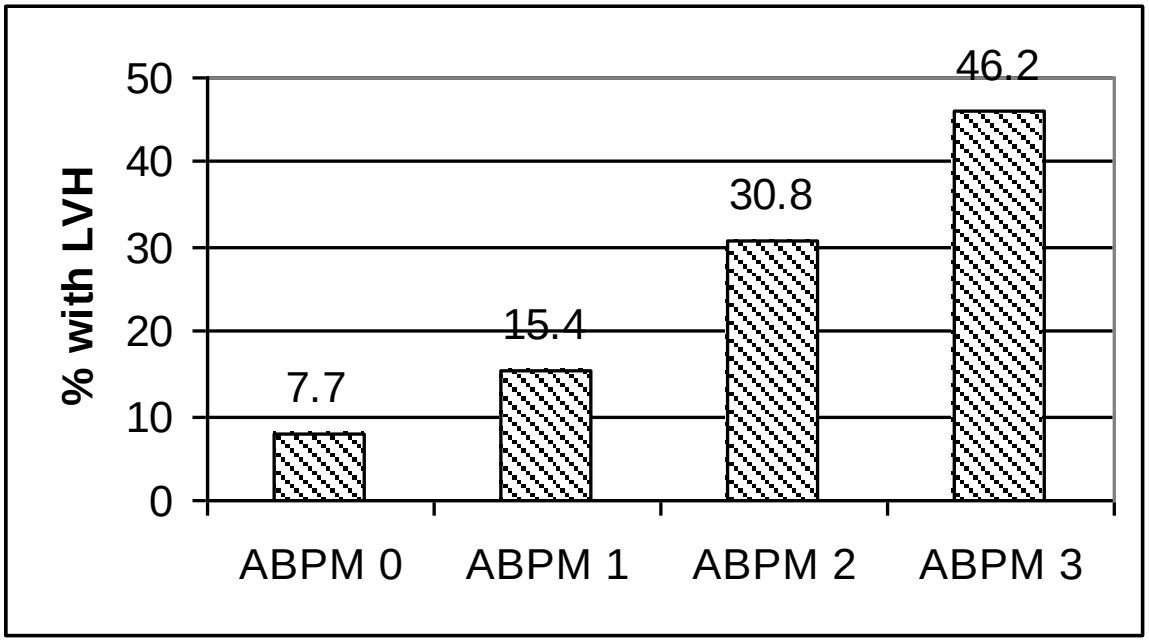
Ambulatory HT is determined using 3 indices: Lack of Nocturnal BP Dipping, BP Load and BP Index. Any one of the indices are adequate to diagnose Ambulatory HT.

We determined the correlation between the number of ABPM Indices and the prevalence of LVH.

Table 22

		ABPM				Total
		0 Indices	**1 Index**	**2 Indices**	**3 Indices**	
LVH	**Absent**	3/33 (9.1%)	17/33 (51.5%)	9/33 (27.3%)	4/33 (12.1%)	33
	Present	1/13 (7.7%)	2/13 (15.4%)	4/13 (30.8%)	6/13 (46.2%)	13
Total		4	19	13	10	46

Figure 22



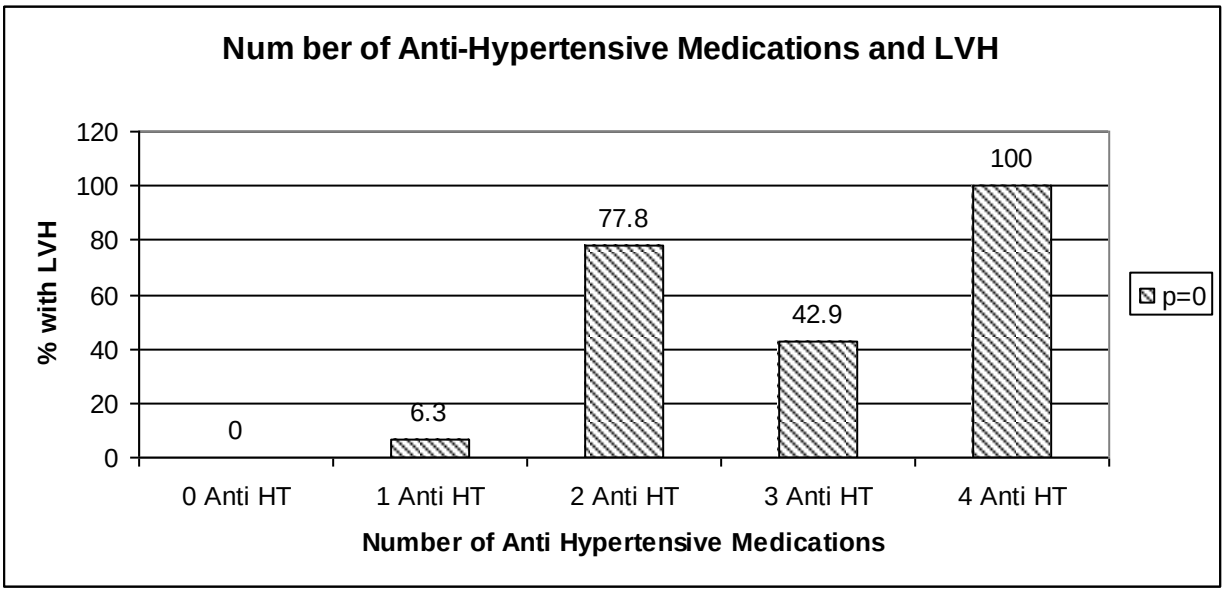
There is significant correlation between the number of ABPM indices present and the Percentage with LVH **(p=0.046).**
When no ABPM index is present, only 7.7% of subjects had LVH.
When 1 ABPM index is present 15.4% of subjects had LVH.
When 2 ABPM indices are present 30.8% of subjects had LVH.
When all 3 ABPM indices are present 46.2% of subjects have LVH.

LVH vs Number of anti hypertensive medications

Table 23

		LVH		Total
		Absent	**Present**	
Number of anti hypertensive medications	**0**	12/12 (100%)	0	12
	1	15/16 (93.8%)	1/16 (6.3%)	16
	2	2/9 (22.2%)	7/9 (77.8%)	9
	3	4/7 (57.1%)	3/7 (42.9%)	7
	4	0	2/2 (100%)	2
Total		33	13	46

Figure 23



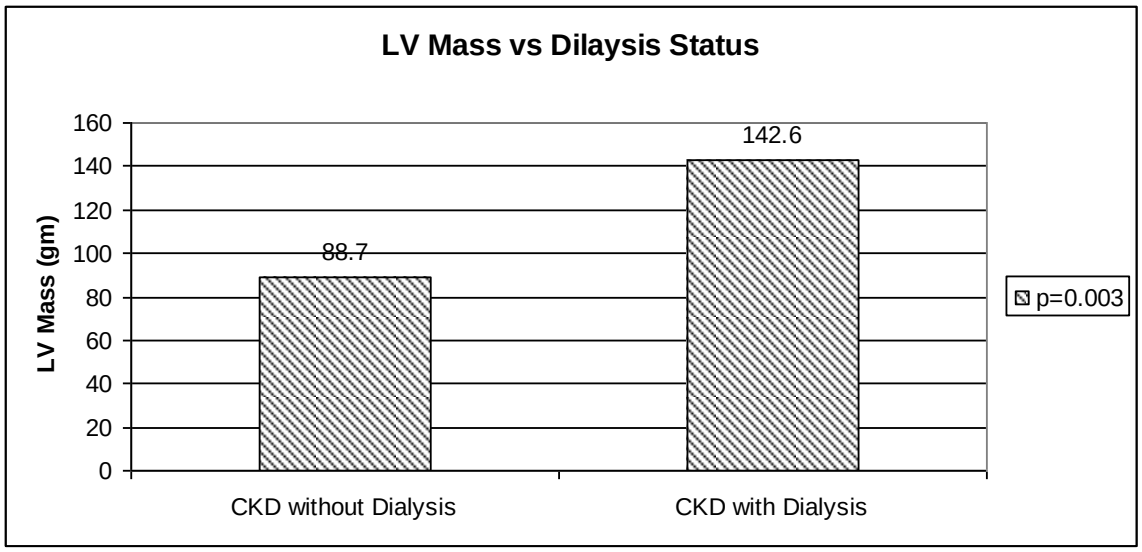
There is a significant correlation between the number of Anti-Hypertensive Medications taken and the percentage of children with LVH **(p=<0.001).**

LV Mass vs Dialysis Status

Table 24

	CKD without Dialysis Mean (SD)	CKD with Dialysis Mean (SD)	P value	Difference between 2 means with 95th % CI
LV Mass	88.7 (45.77)	142.6 (61.9)	0.003	53.9 (19.16-88.7)

Figure 24



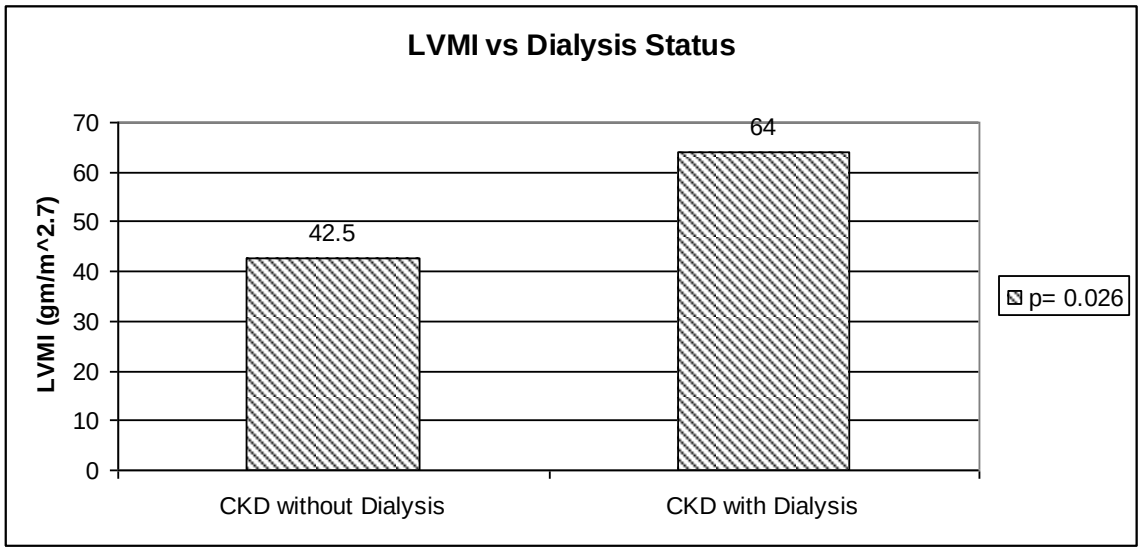
There was a statistically significant difference between the LV Mass of those children with CKD not requiring Dialysis (88.7 gm) and those on Dialysis (142.6 gm **p= 0.003**).

LVMI vs Dialysis Status

Table 25

	CKD without Dialysis Mean (SD)	CKD with Dialysis Mean (SD)	P Value	Difference between 2 means with 95th % CI
LVMI	42.5 (24.6)	64 (34.5)	0.026	21.5 (2.6-40.3)

Figure 25



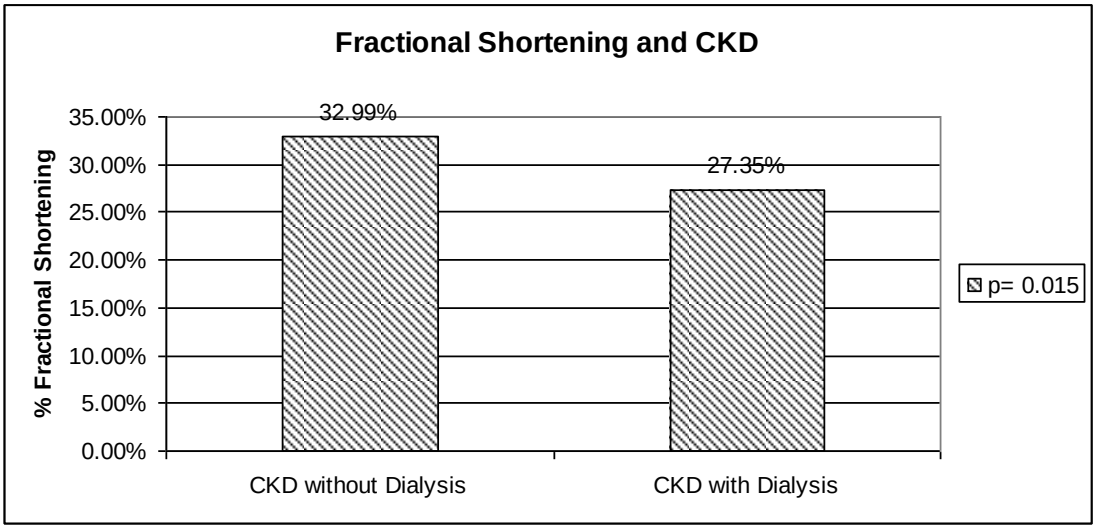
There was a statistically significant difference between the LVMI of those children with CKD not requiring Dialysis (42.5 gm/m^2.7) and those on Dialysis (64 gm/m^2.7 **p= 0.026**).

Fractional Shortening and CKD

Table 26

	CKD without Dialysis Mean (SD)	CKD with Dialysis Mean (SD)	P Value
Fractional Shortening	32.99 (3.55)	27.35 (6.29)	0.015

Figure 26



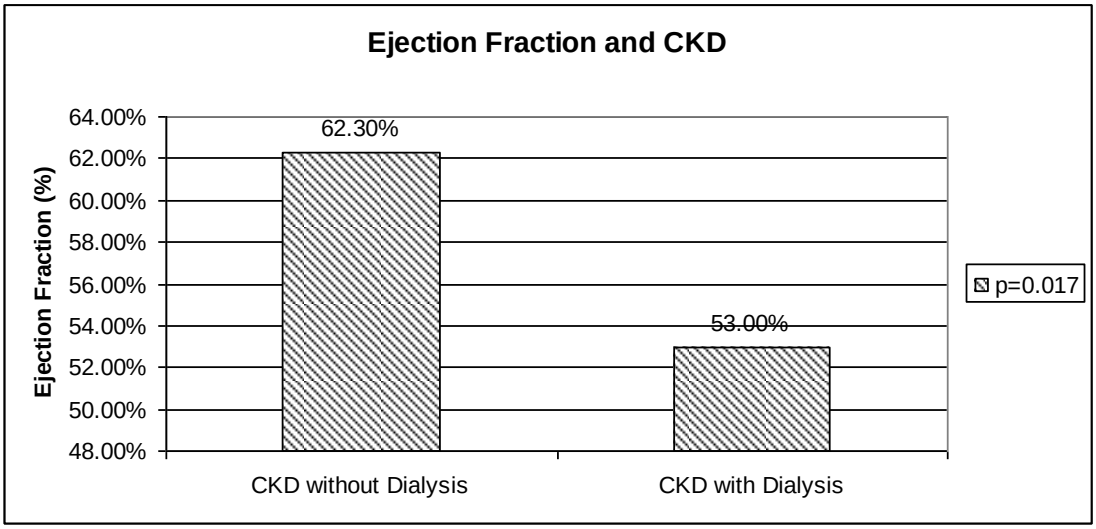
There is a significant difference between the percentage of Fractional Shortening between children with CKD not requiring dialysis 32.99 %, and those on dialysis 27.35% **(p=0.015).**

Ejection Fraction and CKD

Table 27

	CKD without Dialysis Mean (SD)	CKD with Dialysis Mean (SD)	P value
Ejection Fraction (%)	62.3 (4.86)	53.0 (10.73)	0.017

Figure 27



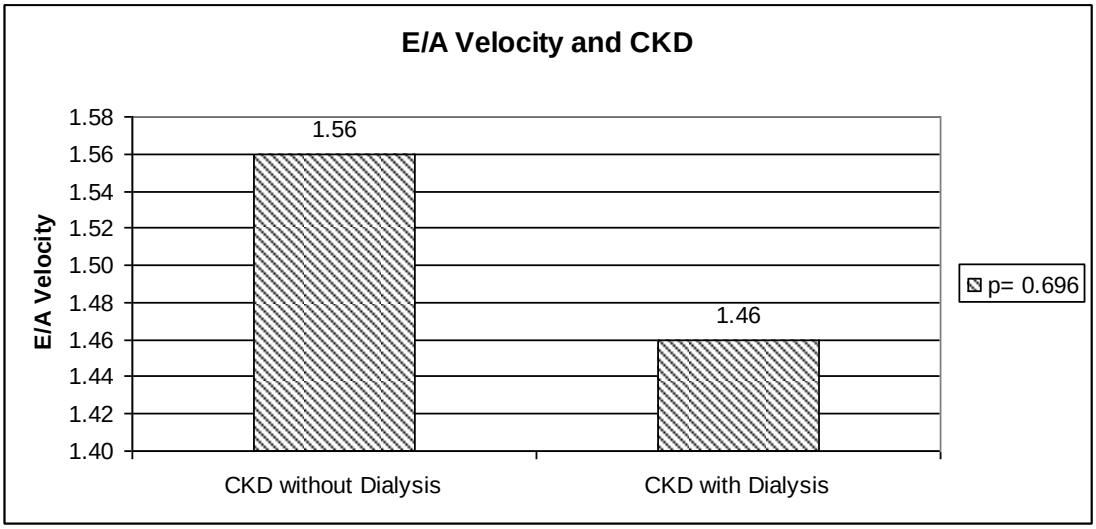
There was a statistically significant difference in the Ejection Fraction between children with CKD not requiring dialysis 62.3% and those on dialysis 53% **(p= 0.017).**

E/A Velocity and CKD

Table 28

	CKD without Dialysis Mean (SD)	CKD with Dialysis Mean (SD)	P Value
E/A Velocity	1.56 (0.27)	1.46 (0.46)	0.696

Figure 28



There was no statistically significant difference in the E/A Velocity amongst children with CKD not requiring Dialysis 1.56, and those on dialysis 1.46 (p=0.696).

Comparison between children with CKD not requiring dialysis and Children on Dialysis

Table 29

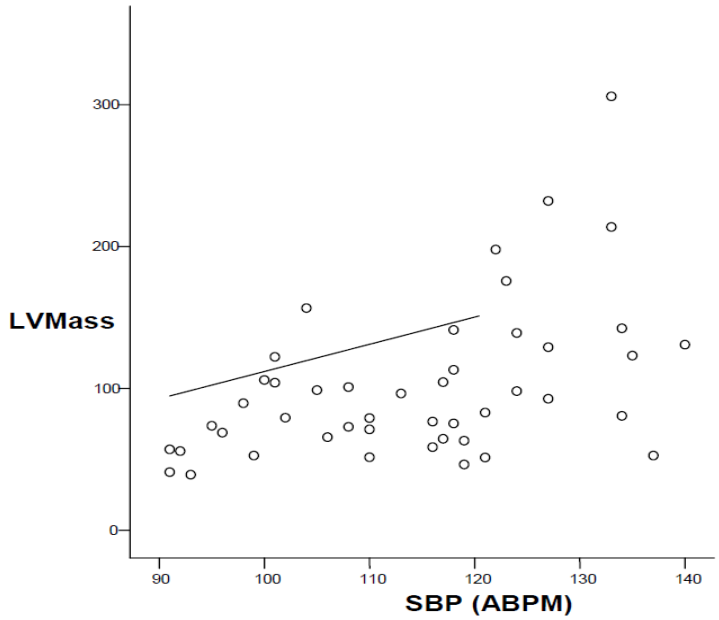
PATIENT CHARACTERISTICS			
PARAMETERS	**CKD without Dialysis Mean (SD) N= 35**	**CKD with Dialysis Mean (SD) N=11**	**P-value**
Age (yrs)	11.26 (3.48)	11.82 (3.03)	0.633
Sex (male)	72.20%	90.00%	0.236
Height (cm)	133.38 (20.01)	137.46 (19.19)	0.554
Weight (kg)	29.81 (11.87)	29.93 (11.69)	0.978
BMI (kg/m^2)	16.14 (3.16)	15.24 (2.33)	0.39
BSA	1.05 (0.27)	1.07 (0.28)	0.795
Stated Duration of CKD (mo)	73.8 (54.31)	27.09 (37.11)	**0.004**
Hypertension (%)	42.9	90.9	**0.005**

Table 30

PATIENT CHARACTERISTICS – MEDICATIONS			
PARAMETERS	**CKD without Dialysis Mean N= 35**	**CKD with Dialysis Mean N=11**	**P-value**
ACEI	57.10%	36.40%	0.196
ARB	11.40%	27.30%	0.207
CCB	28.60%	81.80%	0.003
B-blockers	8.60%	18.20%	0.343
AB	2.90%	36.40%	**0.009**
Vasodilators	2.90%	18.20%	0.138
Calcium	77.10%	81.80%	0.553
Rocaltrol	60.00%	72.70%	0.349
Iron	85.70%	90.90%	0.555
Erythropoietin	22.90%	54.50%	**0.056**
Soda bicarbonate	74.30%	63.60%	0.372

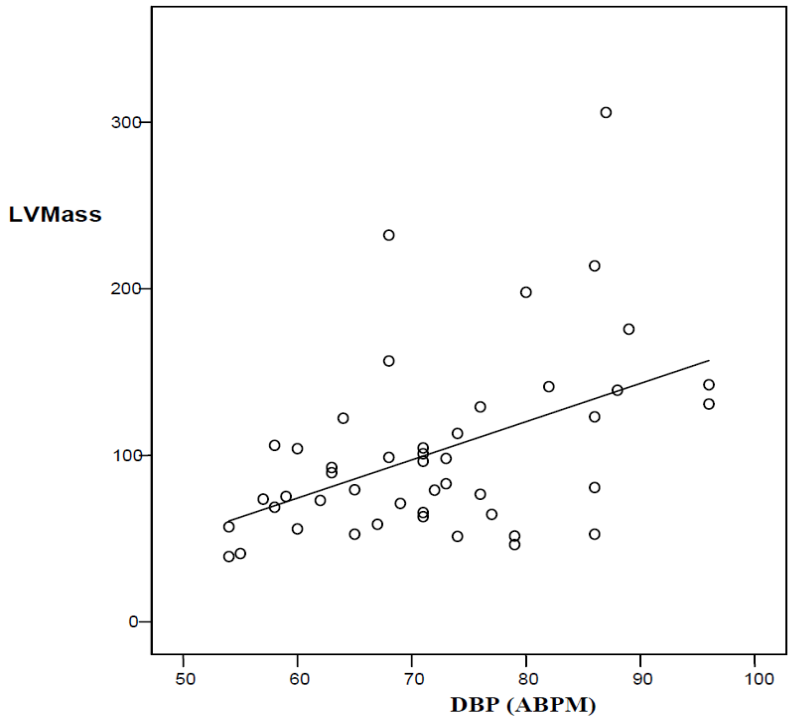
Systolic BP Vs LVMass

The Systolic BP as measured by ABPM was found to correlate linearly with the LVMass (regression coefficient=0.481, **p=0.001).** Figure 31



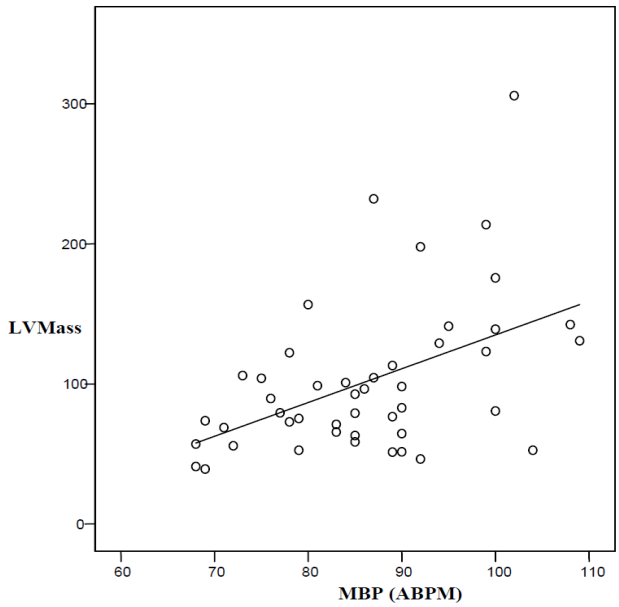
Diastolic BP Vs LVMass

The Diastolic BP as measured by ABPM was found to correlate linearly with the LVMass (regression coefficient=0.467**, p=0.001**). Figure 32



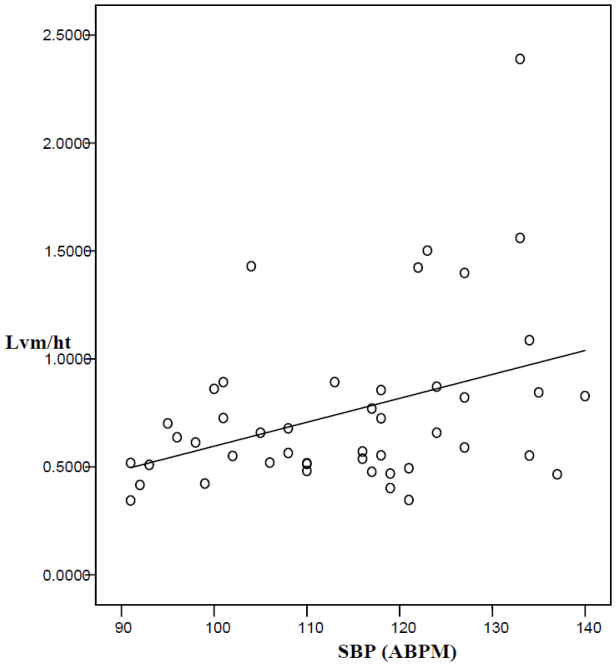
Mean BP Vs LVMass

The Mean BP as measured by ABPM was found to correlate linearly with the LVMass (regression coefficient=0.478, **p=0.001**). Figure 33



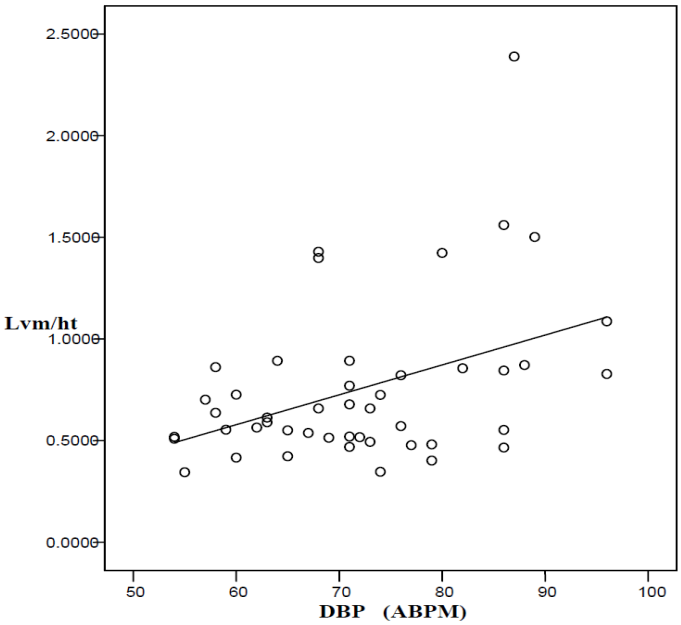
SBP Vs LVM/Ht

The Systolic BP as measured by ABPM was found to correlate linearly with the LVMass/Ht (regression coefficient=0.382, **p=0.009**). Figure 34



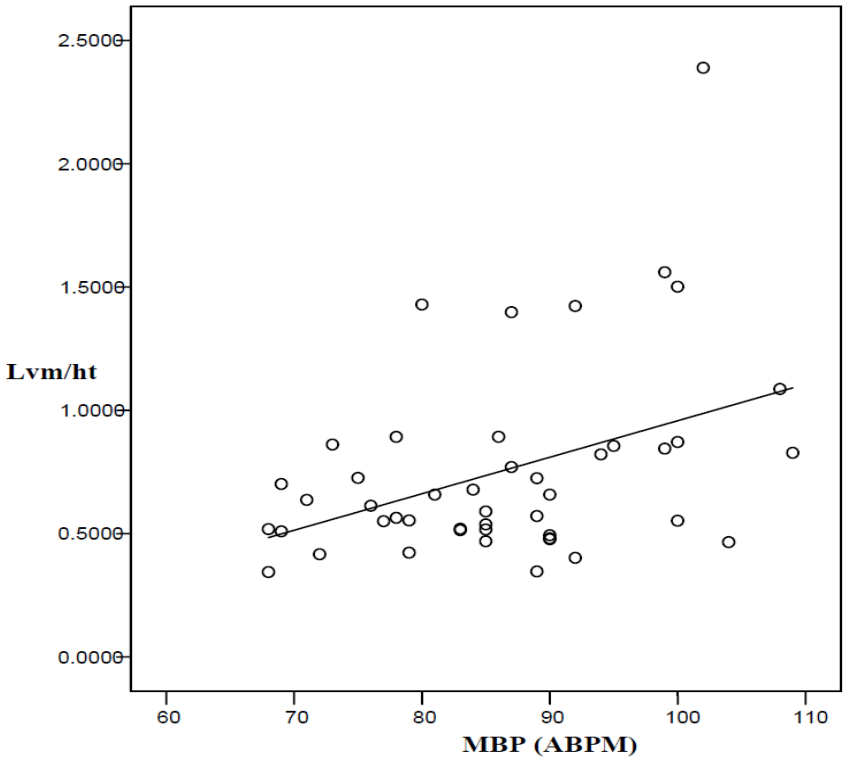
DBP vs LVM/Ht

The Diastolic BP as measured by ABPM was found to correlate linearly with the LVMass/Ht (regression coefficient=0.412, **p=0.004**). Figure 35



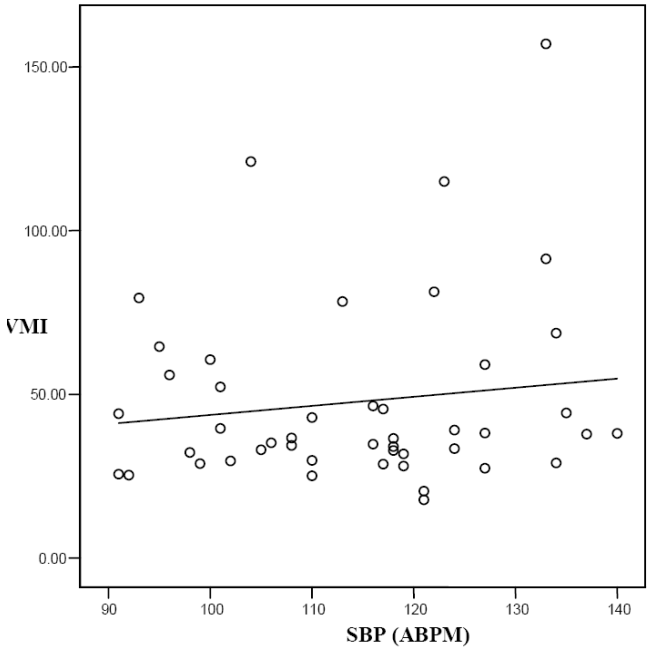
MBP vs LVM/Ht

The Mean BP as measured by ABPM was found to correlate linearly with the LVMass/Ht (regression coefficient=0.403**, p=0.005**). Figure 36



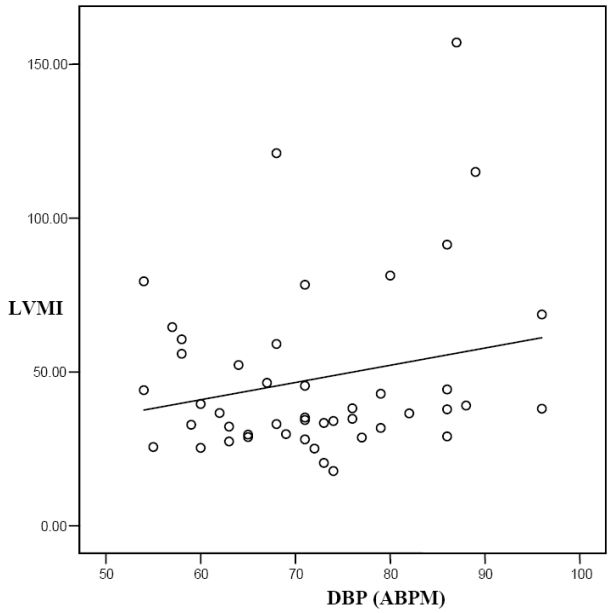
SBP vs LVMI

There was no significant correlation between the Systolic BP as measured by ABPM and the LVMI (regression coefficient=0.018, p=0.375). Figure 37



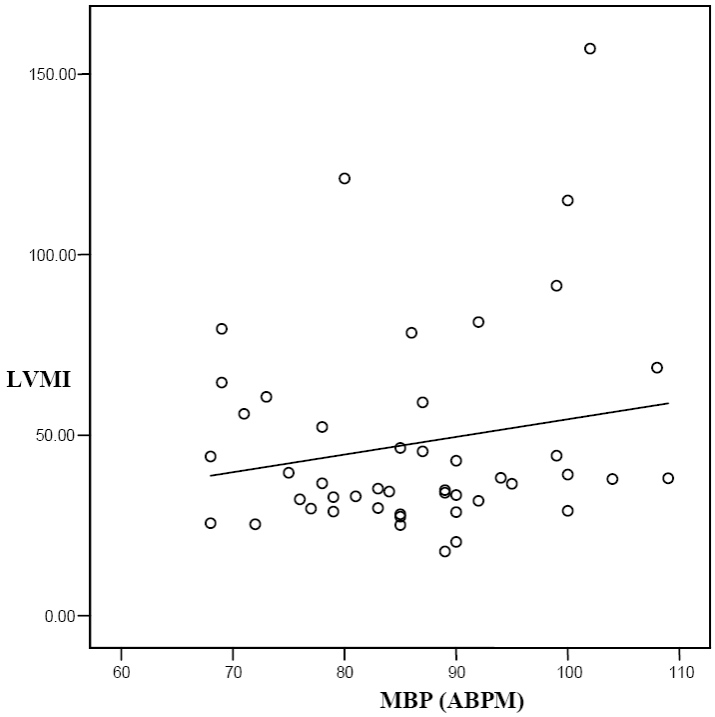
DBP vs LVMI

There was no significant correlation between the Diastolic BP as measured by ABPM and the LVMI (regression coefficient=0.048, p=0.143). Figure 38



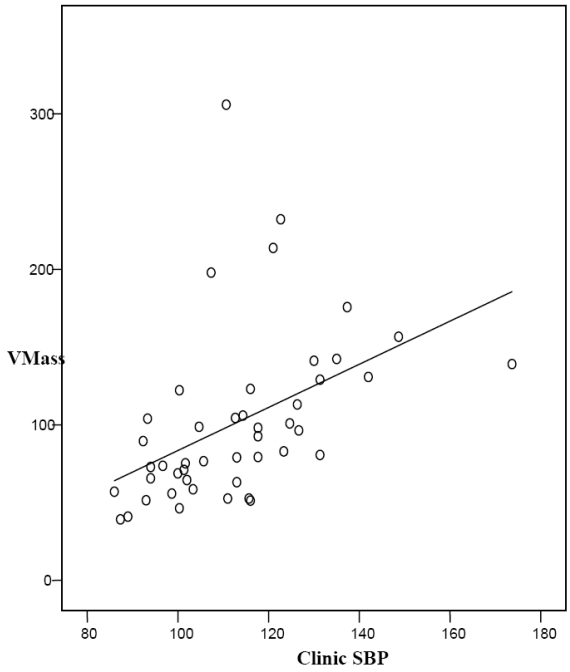
MBP vs LVMI

There was no significant correlation between the Mean BP as measured by ABPM and the LVMI (regression coefficient =0.035, p=0.214). Figure 39



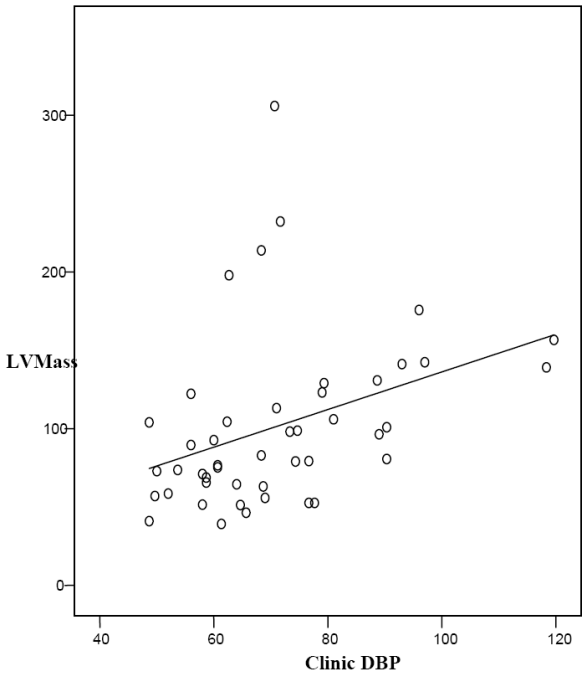
LVMass vs Clinic SBP

The Systolic BP as measured by Clinic Readings was found to correlate linearly with the LVMass (regression coefficient=0.451, p=0.002). Figure 40



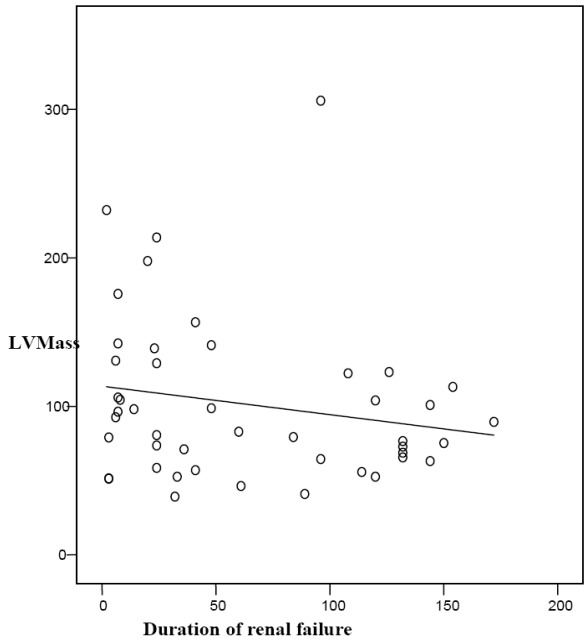
LVMass vs Clinic DBP

The Diastolic BP as measured by Clinic Readings was found to correlate linearly with the LVMass (regression coefficient=0.367, p=0.012). Figure 41



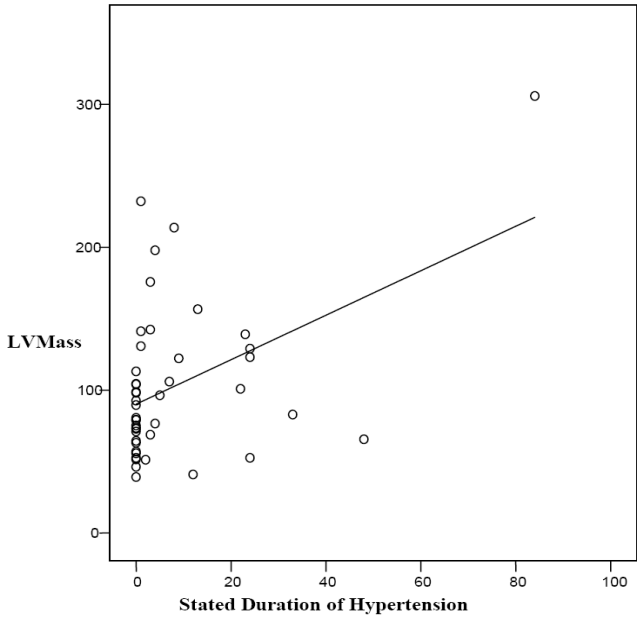
LVM vs Stated Duration of Renal Failure

There was no significant correlation between the stated duration of CKD and the LVMass(regression coefficient= -0.191, p=0.204). Figure 42



LVM vs Stated Duration of Hypertension

There was an apparent linear correlation between the Stated Duration of Hypertension and LVMass (regression coefficient=0.448, p=0.002). Figure 43



Univariate Analysis for variables that impact LVH:

Table 44

Variable	P value	Odds Ratio
BP load	**0.046**	12.687
Anemia	0.253	3.101
BP Index	0.654	0.585
Nocturnal Dipping	0.199	4.749
Clinic BP	**0.030**	8.901
Dialysis	0.866	0.841

The univariate analysis showed that both Clinic BP readings (odds ratio 8.9) and BP Index as measured by ABPM (odds ratio 12.7) had significant contribution towards LVH.

DISCUSSION

DISCUSSION

This study was conducted in the Pediatric Nephrology Unit of Christian Medical College Vellore which is a tertiary referral hospital situated in South India. The study was conducted in the time period from November 2011 to November 2012.

Our study looked at the cardiovascular risk factors amongst children with Chronic Kidney Disease who had an estimated GFR of <60 ml/min/1.73m^2. Children with Chronic Kidney Disease are living longer due to renal replacement therapy in the form of dialysis and renal transplantation. With their increased life spans, they are more prone to developing cardiovascular risks. Many studies have showed that cardiovascular risks are the leading causes of death amongst children with CKD [33,34,76,77]. Thus early identification of cardiovascular risks and their appropriate management can prolong life and prevent mortality.

Forty six children with CKD were recruited in our study. Out of these 37/46 (80.4%) were males, while 9/46 (19.6%) were females (Table 1). The **Male: Female** ratio was found to be 4.1:1. This is similar to reports by Hari et al from AIIMS India who reported 225/305 (73.7%) males in their study [78]. This unequal gender distribution can be explained by the gender selective health care seeking behavior of parents. In India, generally there is a bias towards the male child, thus once detected to have a chronic disease like CKD; parents are more likely to seek health care for male children than for female children.

The **age distribution** of the children was from 3 to 17 years. The maximum number of children was in the age group 12-15 years (17/46 36.9%), followed by 28.3% (13/46) in the 9-12 years age group, 13% (6/46) in the 1-6 years age group and 6-9 years age group and 8.6% (4/46) children > 15 years old (Table 2).

Christian Medical College and Hospital is a tertiary care referral Hospital situated in South India. Amongst our children most were from **India** (44/46 95.6%) while 2/46 (4.3%) were from Bangladesh (Table 3). Amongst all the children from India the maximum number of children were from **Tamil Nadu** (21/46 47.7%) followed by West Bengal (8/46 18.2%) and Andhra Pradesh (6/46 13.6%). The rest of the children were from Jharkhand, Maharashtra, Madhya Pradesh, Odissa, Chattisgarh, Bihar, Assam and Manipur (Table 4).

The commonest **cause of CKD** was found to be Obstructive Uropathy (22/46 48%) including Posterior Urethral Valve, Reflux Nephropathy and Neurogenic Bladder, followed by Dysplastic kidneys (16/48 35%), Glomerular disease (7/46 15%) and Cystic disease (1/46 2%) (Table 5). There were no children with tubular causes of CKD. Data reported by Hari et al in a study done in AIIMS India in 2003 [16] showed similar results with Obstructive Uropathy was the commonest cause (31.8%) followed by Chronic Glomerulonephritis (27.5%) and Reflux Nephropathy (16.7%).

The children were classified into various **stages of CKD** according to the KDOQI classification [2]. According to the aim of our study only children with stage 3, 4 and 5 CKD were included in our study. There were 39.1% (18/46) children in both stage 3 and 4 CKD while there were 21.7 % (10/46) children with stage 5 CKD or undergoing dialysis (Table 6).

For further analysis all children with CKD were divided into two groups as shown in Table 8:

1. Those with CKD not requiring Dialysis (35/46 76%)
2. Those with CKD with Dialysis (11/46 23.9%)

Amongst the 11 children on Dialysis 7/11 (63.6%) were on Continuous Ambulatory Peritoneal Dialysis while 4/11 (36.4%) were on Hemodialysis.

The **complications of children on CKD** were compared between the two groups.

Out of 46 children, 42 (93.1%) were found to have at least one cardiovascular risk factor.

As part of the analysis of cardiovascular risks in children with CKD the nutritional status of children in the two groups was compared using BMI as an index. Our data showed low BMI in children when compared to other studies. Nutritional status of children with CKD is often suboptimal due to metabolic acidosis, uremia, nausea and ongoing chronic inflammation. Despite dietary counseling and regular follow up, most children had poor nutritional status. Despite adequate dialysis the nutrition of our children did not improve significantly. This could be due to the late disease presentation.

The **Body Mass Index** of children as compared to age and sex matched controls was found to be low in (Table 7). This is similar to the study done by D G Noone et al in which the BMI correlated with the degree of renal failure, with children with more advanced CKD having lower BMI [79]. We did not find any statistically significant differences in the Body Mass Index amongst children with CKD without dialysis and those undergoing dialysis.

There were 11/46 children on dialysis, while 35/46 children were on conservative management for CKD (Table 8).

In other studies, the Calcium-Phosphorous Product has been found to be a marker of Carotid Intimal Medial thickness and thereby an early marker of atherosclerosis. In our study we did not find any Statistically significant difference in the Calcium-Phosphorus Product between the two groups, though the dialysis group had higher Ca X PO4 product (p= 0.227) (Table 9).

The prevalence of **Anemia** was found to be higher amongst those on Dialysis (8/11 72.7%) compared to the non-dialysis group (17/35 48.2%). But this difference was not statistically significant (p= 0.145) (Table 10). This was in keeping with findings from Wong et al who found 31% of children with stage 1 CKD to have anemia while 93.3% of those with stage 4 and 5 CKD had anemia (p=<0.01) [17.]

Erythropoietin was used in 14/46 (30.4%) children studied. Out of these 6/14 (42.8%) children were on dialysis. 64.3% (9/14) children on erythropoietin were still anemic.

The **electrolyte status** was assessed in all children. The main parameter considered was **Hyperkalemia** since most children had sodium values in the normal range. Hyperkalemia was detected in 18.2% (2/11) of children on dialysis compared to 17.1% (6/35) in non-dialysis children **(p= 0.026)** (Table 11). Wong et al have reported much lower rates of hyperkalemia with 5.26% children with stage 3 CKD and 18.18 % children with stage 4 and 5 CKD found to have hyperkalemia [17]. According to Alcazar et al the causes of hyperkalemia were drugs (ACEI, ARB, NSAIDS, and Heparin), constipation, prolonged fasting and metabolic acidosis [22]. The causes of hyperkalemia were not elaborated in our patients, but may have been due to dietary factors and usage of ACEI.

The prevalence of **Metabolic Acidosis** as defined by serum Bicarbonate levels below 20 mg/dl was found to be more amongst children not requiring dialysis (21/35 60%) compared to those on dialysis (6/11 54.4%) but it was not statistically significant (p= 0.508) (Table 12). In advanced stages of CKD these results matched those of Wong et al, with CKD stage 4 and 5 having 54.55% metabolic acidosis [17]. Wong reported much lower rates of metabolic acidosis amongst children with stage 3 CKD (7.89%).

The phosphorus status is related to the dietary intake of children. There was a statistically significant difference in the prevalence of **Hyperphosphatemia** amongst the non-dialysis group (7/34 20.6%) and those on dialysis (7/10 70% **p= 0.006**) (Table 13). This is despite strict dietary restrictions and treatment with Phosphate binders.

Metabolic Bone Disease is am important complication of CKD. If poorly controlled, morbidity can be high due to renal osteodystrophy. The prevalence of **Metabolic Bone Disease** as defined by a serum Parathyroid hormone level >250 pg/dl was compared between the non-dialysis group (12/35 34.3%) and those on dialysis (7/10 70% **p=0.05**) which was found to be statistically significant (Table 14). Similar results were demonstrated by Wong et al who showed that the prevalence of metabolic bone disease was 43.3% in stage 3 CKD and 100 % in stage 4 and 5 CKD [17].

The prevalence of **Proteinuria** was higher amongst the non-dialysis group (18/20 90%) compared to those on dialysis (2/3 66.6%) but it was not statistically significant (p= 0.356) (Table 15). Wong et al defined proteinuria as urinary protein excretion greater than 1 gm/day. They found the prevalence of proteinuria to be 5.6% in stage 1 CKD and upto 40 % in stage 4 and 5 CKD [17]. Though the percentage of proteinuria amongst those on dialysis was less, the severity of proteinuria was greater in those on dialysis.

The aim of our study was to determine the cardiovascular risk factors amongst children with CKD. The cardiovascular risks were determined using **clinic BP readings, ABPM and ECHO.** ABPM is not widely used in India to detect hypertension even though it's the gold standard for detecting hypertension [39]. There are 3 main indices used to assess BP by ABPM. The **BP load** tells about the percentage of BP readings above the 95th centile for age, sex and height. The **BP Index** is the average BP for each patient divided by the 95th centile BP value specific for that

patient. **Nocturnal dipping** indicated the normal fall in BP during night time which should be > 10% [39].

The prevalence of **Hypertension** was determined using both **Clinic BP readings** and with 24 hour **Ambulatory BP Monitoring** (Table 16). Clinic Hypertension was detected in 10/46 (21.7%) children. Using ABPM as the gold standard for detecting hypertension 91.3 % of children studied were found to be hypertensive (Table). This is in keeping with reports from Wong et al who found the prevalence of HT to be 63.29% in stage 1 CKD and upto 80 % in stage 4 and 5 CKD [17]. Using ABPM a total of 42/46 (91.3%) of children with CKD were found to be hypertensive.

An additional 32 children were found to have Masked Hypertension using ABPM. Children with masked hypertension cannot be picked up by Clinic BP readings alone. In a more recent study done by Mark Mitsnefes et al who found 38% children had masked HT [24]. He also showed that 34% of children with confirmed HT had LVH while 20% of children with masked HT had LVH. By extrapolation routine BP recording in the Out Patient setting is missing upto 76.2% of HT in children with CKD.

Hypertension is a very important cardiovascular risk. It's also highly amenable to treatment. Thus early detection of hypertension and adequate control are paramount to preventing progression of cardiovascular risks.

The prevalence of Hypertension was compared with the class of CKD. Our study showed that **Hypertension** increases with worsening classes of CKD (Table 17). In Stage 3 CKD **clinic HT** was

present in 4/18 (22.2%) while **ambulatory HT** was present in 15/18 (83.3%) children. Amongst children with Class 4 CKD only 5.5% (1/18) had Clinic HT as against 94.4% (17/18) with Ambulatory HT. While those with Class 5 CKD 5/10 (50%) were detected to had Clinic Hypertension and all 10 (100%) had hypertension by ABPM. According to Mark Mitsnefes et al also the prevalence of hypertension amongst children on dialysis is as high as 52-75% [3].

Despite normal Clinic BP readings ABPM still detects hypertension suggesting inadequate BP control. This goes to show that clinic BP readings are not sufficient to assess hypertension amongst children with CKD.

16.6% (2/12) children with **Clinic Hypertension** were not on any **anti hypertensive medications** and had newly detected HT. While 8/34 (23.5%) were on anti hypertensive medications, but with inadequate BP control (Table 18).

Out of the children who were diagnosed to have **Ambulatory HT** 12/12 (100%) were not on any **anti hypertensive medications** and were newly detected to have hypertension. Whereas 30/34 (88.2%) were on anti hypertensive medications but had inadequate BP control (Table 19).

Erythropoietin is used amongst children with CKD to prevent anemia and reduce the number of blood transfusions. One of the known side effects of erythropoietin is hypertension. We looked at all the children with CKD who received treatment with Erythropoietin and compared with blood pressures with those not receiving erythropoietin. Children on treatment with erythropoietin were found to have greater **SBP, DBP and MBP** compared with those not receiving erythropoietin (Table 20). One confounder

could be that children with higher stages of CKD are likely to have more anemia and thus prescribed erythropoietin. These children also have a greater chance of being hypertensive in view of more advanced stage of CKD. Thus it is difficult to ascertain if the hypertension is secondary to erythropoietin usage or just part of the natural course of CKD.

ECHO was used to determine LV Dysfunction amongst all children with CKD. The LV Mass was calculated using the Devereux formula [54] :

LVM (g) = 0.8 {1.04 [(*LVEDD* + *PWT* +*IVST*)3 -(*LVEDD*)3] + 0.6}

LV Hypertrophy was defined as LV Mass greater than 95th centile for age and sex [56]. Children with Dialysis had a significantly higher percentage of LVH (6/11 54.4%) compared to children without dialysis (7/35 20% **p= 0.036**) (Table 21).

Hypertension is a major risk factor for cardiovascular disease amongst children with CKD. ABPM is the gold standard to determine hypertension. As more than one ABPM indices were taken into account, the prevalence of **LVH** increased (Table 22). When only 1out of 3 ABPM indices was present 2/13 (15.4%) had LVH, when 2 indices were present 4/13 (3.8%) and when all 3 indices were present 6/13 (46.2%) have LVH. This correlation was found to be statistically significant **(p= 0.046).**

A literature search did not reveal any definite guidelines as to how many BP Indices need to be positive to indicate hypertension using ABPM. We need further studies in this field to correlate hypertension as defined by ABPM with LVH and to see at what exact cutoff LVH occurs. This

would help us define at what BP levels LVH is likely to set in and preventing this would be a target for adequacy of hypertensive control. ABPM is considered the most predictive of end organ damage [39].

In our study we also found a significant correlation between the **number of anti hypertensive medications** used and the prevalence of **LVH (p=<0.001)**. Children on 1 anti-HT had 6.3% chance of LVH, those on 2 anti-HT medications had 77.8%, those of 3 anti-HT medications had 42.9% and those on 4 anti-HT medications had 100% chance of having LVH (Table 23). This emphasizes the importance of adequate hypertension control to prevent LVH.

The **LV Mass** was compared between the non-dialysis group and children on dialysis. There was a statistically significant difference between the LV Mass of those children with CKD not requiring Dialysis (88.7 gm) and those on Dialysis (142.6 gm **p= 0.003**) (Table 24).

The **LVMass Index** was compared between the two groups. The children undergoing dialysis were found to have a significantly higher LVMI (64 gm/m^2.7) compared to children in the non-dialysis group (42.5 gm/m^2.7) (**p= 0.026**) (Table 25).

The percentage of **Fractional Shortening** was found to be significantly less in the dialysis group (27.35%) compared to the non-dialysis group (32.99 %) **(p=0.015)** (Table 26).
There was a statistically significant difference in the **Ejection Fraction** between children with CKD not requiring dialysis 62.3% and those on dialysis 53% **(p= 0.017)** (Table 27).

There was no statistically significant difference in the **E/A Velocity** amongst children with CKD not requiring Dialysis 1.56, and those on dialysis 1.46 (p=0.696) (Table 28).

The ABPM readings for **SBP, DBP and MBP** were found to very significantly correlate with the **LV Mass (p= 0.001)** (Figure 31-33). The SBP, DBP and MBP readings also correlated with **LVMass/ Height**, but to a lesser extent than with LV Mass alone (**p= 0.009, p=0.004, p=0.005** respectively) (Figure 34-36). The SBP, DBP and MBP were not found to correlate with the **LVM Index** (p=0.375, p=0.143 and p=0.214 respectively) (Figure 37-39). The **Clinic SBP and DBP** readings also correlated with the LVMass but to a lesser degree than the ABPM reading (**p= 0.002 and p= 0.012** respectively) (Figure 40, 41). This leads on to show that ABPM is a better assessment of BP than clinic BP readings alone.

It was postulated that longer duration of CKD would lead to greater cardiovascular risks in children. But the **LV mass** was not found to correlate with the **Stated Duration of CKD** (regression coefficient= -0.191, p=0.204) (Figure 42). This could be explained by the time gap between disease onset and when symptoms set it. Due to a lack of regular ante-natal screening for urinary tract anomalies or any routine screening of school children for CKD, children with CKD present late to medical attention when complications of CKD like anemia, metabolic bone disease, growth failure, fluid overload and acidosis have already set in. Thus there is a significant time lag between the onset of disease and when it is detected. This could probably explain the lack of correlation between stated duration of CKD and LVMass.

If we were able to determine the true duration of CKD, it would probable correlate with degree of LVH.

There is an apparent linear correlation between the **Stated Duration of Hypertension** and **LVMass** (regression coefficient=0.448, **p=0.002**) (Figure 43). The longer the children have Hypertension the more they are predisposed to have LV overload and subsequent LVH.

In a univariate analysis done to determine the factors contributing to LVH we found that only **BP Load** and **Clinic BP** were found to be statistically significant (**p= 0.046 and p=0.030** respectively) (Table 44). Children with significant BP Load had 12.7 times greater chance of having LVH. Children with Clinic HT had 8.9 times greater chance of having LVH. Lack of **Nocturnal Dipping** led to a 4.8 time greater chance of having LVH, while **Anemia** led to a 3.1 times greater chance of having LVH.

Amongst our children studied two children on dialysis died during our study period. Both children died at home and the exact cause of death could not be determined accurately. Their deaths too could have been due to cardiovascular causes.

From our study we have learnt that regular ABPM and ECHO may be necessary in reducing the cardiovascular morbidity and mortality amongst children with CKD. We need to use these test as part of our routine practice to help our children with CKD to live longer and healthier lives.

SUMMARY

SUMMARY

1. This study was conducted in the Pediatric Nephrology Unit of Christian Medical College Vellore which is a tertiary referral hospital situated in South India.
2. A total of 46 children with CKD were studies.
3. The Male: Female Ratio was found to be 4.1:1.
4. The age distribution of the children was from 3 to 17 years. The maximum number of children was in the age group 12-15 years (17/46 36.9%).
5. Of the 46 children with CKD studied, 44/46 (96%) were from India. 2/46 (4%) of the children were from Bangladesh.
6. Obstructive Uropathy was the commonest cause of CKD 22/46 (48%) followed by Dysplastic kidneys16/46 (35%).
7. Of the 46 children with CKD, 18/46 (39.1%) were in CKD Stage 3 and 4 respectively, while 10/46 (21.7%) had CKD Stage 5 or were on Dialysis.
8. The children were divided into two groups for the purpose of analysis:

   Children with CKD not requiring Dialysis

   Children with CKD on Dialysis
9. Amongst the non-dialysis group 28.6 %(10/35) had normal BMI, while 40% (14/35) had BMI +/- 2 Z scores, 28.6 % (10/35) had BMI +/- 3 Z scores while 2.9 % (1/35) had BMI +/- 4 Z scores. Amongst children on Dialysis 18.2 5 (2/11) had normal BMI, while 36.4 % (4/11) had BMI +/- 2 Z scores, 45.5 % (5/11) had BMI +/- 3 Z scores.

Amongst children on Dialysis 18.2 5 (2/11) had normal BMI, while 36.4 % (4/11) had BMI +/- 2 Z scores, 45.5 % (5/11) had BMI +/- 3 Z scores

10. The calcium phosphorus product was found to be elevated in children with dialysis but it was not statistically significant compared to the non-dialysis group (p=0.227).
11. There was no significant difference in the prevalence of anemia amongst children with CKD without Dialysis 17/35 (48.6%) and children on dialysis 8/11 (72.7%) (p=0.145).
12. There was significant difference in the prevalence of Hyperkalemia amongst children with CKD not requiring dialysis 6/35 (17.1%) and children on dialysis 2/11 (18.2%) **(p=0.026).**
13. There was no significant difference in the prevalence of Metabolic Acidosis between children with CKD not requiring dialysis 21/35 (60%) and children on dialysis 6/11 (54.4%) (p=0.508).
14. There was a significant difference in the prevalence of Hyperphosphatemia amongst children with CKD not requiring dialysis 7/34 (20.6%) and children on dialysis 7/10 (70%) **(p=0.006).**
15. Children on dialysis had a greater prevalence of Metabolic Bone Disease 7/10 (70%) as compared to children with CKD not requiring dialysis12/35 (34.3%) **(p=0.05).**
16. There was no significant difference in the prevalence of Proteinuria between children with CKD not requiring dialysis 18/20 (90%) and children on dialysis 2/3 (66.6%) (p=0.356).
17. Using Clinic BP alone 10/46 (21.7%) of children with CKD were found to be hypertensive.

18. With the additional usage of ABPM 42/46 (91.3%) of children with CKD were found to be hypertensive, an additional 32/46 (69.5%) were found to have masked hypertension.
19. Out of the children diagnosed to have Clinic Hypertension, 2/12 (16.6%) were not on any anti hypertensive medications, while 8/34 (23.5%) were on anti hypertensive medications.
20. There was no significant difference between the prevalence of Ambulatory HT amongst those not on anti HT medications 12/12 (100%) or users of anti HT medications 30/34 (88.2%) (p= 0.284).
21. There was a significant difference in the prevalence of LVH amongst children on dialysis 6/11 (54.4%) and children with CKD not requiring dialysis 7/35 (20%) (p=0.036).
22. There is significant correlation between the number of ABPM indices present and the Percentage with LVH **(p=0.046).**
23. The Systolic BP, Diastolic BP and Mean BP was found to be higher amongst those children on Erythropoietin when compared to children not using Erythropoietin.
24. There was a significant difference in the prevalence of LVH amongst children on dialysis 6/11 (54.4%) and children with CKD not requiring dialysis 7/35 (20%) **(p=0.036).**
25. There is a significant correlation between the number of Anti-Hypertensive Medications taken and the percentage of children with LVH **(p=< 0.001).**

    Children on 1 anti-HT had 6.3% chance of LVH, those on 2 anti-HT medications had 77.8% chance of LVH, those of 3 anti-HT medications had 42.9% chance of LVH and 100% of children using 4 anti-HT medications had LVH.
26. There was a statistically significant difference between the LV Mass of those children with CKD not requiring Dialysis (88.7 gm) and those on Dialysis (142.6 gm **p= 0.003**).

27. There was a statistically significant difference between the LVMI of those children with CKD not requiring Dialysis (42.5 gm/m^2.7) and those on Dialysis (64 gm/m^2.7 **p= 0.026**).
28. There is a significant difference between the percentage of Fractional Shortening between children with CKD not requiring dialysis 32.99 %, and those on dialysis 27.35% **(p=0.015).**
29. There was a statistically significant difference in the Ejection Fraction between children with CKD not requiring dialysis 62.3% and those on dialysis 53% **(p= 0.017).**
30. There was no statistically significant difference in the E/A Velocity amongst children with CKD not requiring Dialysis 1.56, and those on dialysis 1.46 (p=0.696).
31. The Systolic BP, Diastolic BP and Mean BP as measured by ABPM were found to correlate linearly with the LVMass **(p=0.001).**
32. The Systolic BP, Diastolic BP and Mean BP as measured by ABPM were found to correlate linearly with the LVMass/ Ht (**p=0.009, p= 0.004, p= 0.005** respectively).
33. The Systolic BP, Diastolic BP and Mean BP as measured by ABPM were found to correlate linearly with the LVMass/ Ht^2.7 (p= 0.375, p=0.143 and p=0.214 respectively).
34. The Systolic BP and Diastolic BP as measured by Clinic Readings were found to correlate linearly with the LVMass (**p=0.002, p= 0.012** respectively).
35. There was no significant correlation between the stated duration of CKD and the LVMass (regression coefficient= -0.191, p=0.204).
36. There was an apparent linear correlation between the Stated Duration of Hypertension and LVMass (regression coefficient=0.448, **p=0.002**).

37. In a Univariate analysis done to determine the indices which were the best predictors of LVH, BP Load and Clinic BP were the most significant (Odds ratio 12.7 and 8.9 respectively).

CONCLUSIONS

CONCLUSIONS

1. Children with CKD are at very high risk for cardiovascular morbidity (Hypertension and Left Ventricular Dysfunction).

2. Masked Hypertension is common amongst children with CKD. As clinic BP recordings alone cannot detect masked hypertension; ABPM is a very useful tool.

3. LV Dysfunction is also common in children with CKD.

4. Clinic BP readings and BP Index (by ABPM) are significant predictors of LV Hypertrophy and important tools to monitor cardiovascular dysfunction.

5. L V Mass which correlates well with Systolic, Diastolic and Mean BP readings is an indicator of adequacy of BP control.

LIMITATIONS

LIMITATIONS

1. Normative values for Left Ventricular Mass or ABPM readings according to age, gender and height are not available for Indian children. Western standards used may not be representative of our population.
2. Carotid Intimal Thickness could not be assessed as specific equipment was not available in our institution.
3. Dyslipidemia which is an important risk factor for early atherosclerosis and cardiovascular risk was not assessed.

RECOMMENDATIONS

RECOMMENDATIONS

1. All children with CKD should have an ABPM recording to detect hypertension and ECHO to detect cardiovascular risks at presentation and follow up thereafter.
2. Strict BP control and early interventions to improve Left Ventricular Dysfunction should be undertaken to reduce morbidity and mortality.
3. Our study design was a cross sectional study amongst children with CKD. More longitudinal studies are needed to determine cardiovascular risks with the progression of CKD.

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ANNEXURE I

BIBLIOGRAPHY

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ANNEXURES

ANNEXURE II

CLINICAL RESEARCH FORM

CARDIOVASCULAR MORBIDITY IN CHILDREN WITH CHRONIC RENAL DISEASE STUDY

NAME: HOSPITAL NO.:
AGE: DATE OF BIRTH:
GENDER:ADDRESS:
PHONE NUMBER:

HISTORY:

CAUSE OF CRF (SPECIFY):
1 Cystic disease
2 Glomerular
3 Tubular
4 Dysplastic
5 Uropathy

DURATION OF CRF:

CLASS OF CRF WITH GRF:

DIALYSIS: YES/NO

DURATION OF DIALYSIS:

HYPERTENSION: PRESENT ( ) ABSENT ( )
DURATION:
NUMBER OF ANTI HYPERTENSIVE DRUGS:
WELL CONTROLLED: YES ( ) NO ( )

CURRENT MEDICATIONS:

ANTIHYPERTENSIVES:
1 ACE Inhibitors
2 Angiotensin Receptor Blockers
3 Calcium Channel Blockers
4 a Antagonists
5 Others
CALCIUM:
CALCITRIOL:
PHOSPHATE BINDERS:
IRON:
ERYTHROPOIETIN:
STATINS:
BICARBONATE:
ROCALTROL:
OTHERS:

EXAMINATION:

WEIGHT:

HEIGHT:

BODY MASS INDEX:

BODY SURFACE AREA:

ODEMA CLASSIFICATION:

CLINICAL BP (3 READINGS):

AMBULATORY BP MONITORING (24 HRS):

INVESTIGATIONS:

HEMOGLOBIN:
SODIUM:
POTASSIUM:
BICARBONATE:
CALCIUM:
PHOSPHORUS:
CREATININE:
UREA:
PARATHYROID HORMONE
LIPID PROFILE:
Cholesterol:
TAG:
HDL:
LDL:
URINE ROUTINUE:
PROTEINURIA:
URINE PROTEIN/CREATININE RATIO:
VITAMIN D LEVELS:
TRANSFERRIN SATURATION/ TOTAL IRON BINDING CAPASITY:
CALCIUM x PHOSPHATE PRODUCT:

CAROTID INTIMAL THICKNESS BY USG DOPPLER:

ECHO PARAMETERS:
LV Mass:
LVM Index: $\frac{\text{LVM}}{\text{Height}}$
Relative Wall Thickness:
LV Hypertrophy: Concentric
Eccentric
Normal LV Geometry:
LV Diastolic action:

ANNEXURE III

INFORMED CONSENT FORM

PATIENT INFORMATION SHEET

PEDIATRIC NEPHROLOGY UNIT
CHRISTIAN MEDICAL COLLEGE AND HOSPITAL VELLORE

Title of Research: To study the cardiovascular consequences in children with Chronic Kidney Disease

You are being requested to allow your child to participate in a study to see if they have any increased cardiovascular risks due to Chronic Kidney Disease.

What is the need for the study?
Chronic Kidney Disease affects children world wide. These children have hypertension, heart failure and abnormal lipid profiles, all of which increase their cardiovascular risks. In children with CKD 40% of the deaths are due to cardiovascular causes. By checking Blood Pressure, Carotid USG Doppler, ECHO testing we hope to detect these cardiovascular risks amongst your children early, thereby preventing cardiac morbidity and mortality.

Is there any harm to your child in participating in the study?
We plan to do additional tests in all children:
1 24 hours Ambulatory Blood Pressure Monitoring: This is a simple test which checks Blood Pressure over 24 hours. It does not cause any pain or discomfort to the child.
2 ECHO: This is a painless, non invasive test which tells us about the functioning of the heart and early onset heart failure.
3 USG Doppler of Carotid Intimal Thickness: This is a USG Doppler test of the neck veins to detect any atherosclerosis.
All these tests are non-invasive tests and do not cause any pain or radiation exposure to your child. These tests are recommended in all children with CKD, but are not being done on a routine basis as yet.

If your child takes part, what will be done?
If you give consent for your child's participation in the study we will enter the details about your child's disease into a Performa. In addition to the routine blood tests, we will do three new tests. We will measure the 24 hours Ambulatory Blood Pressure, ECHO and USG Doppler of the Carotids to determine Intimal Thickness. All other treatments that your child is already receiving will be continued.

Can your child withdraw from this study after it starts?
Your child's participation in this study is entirely voluntary and you are also free to decide to withdraw permission to allow your child's participation at any time during the study. If you do so, this will not affect your usual treatment in the hospital in any way.

Will you have to pay for the study tests?
The 24 hours Ambulatory Blood Pressure Monitoring, ECHO and USG Doppler of Carotids for Intimal thickness will be done free of cost. Any other treatment that your child usually takes will continue and the routine charges for the treatment will be paid by you.

Will your personal details be kept confidential?
The results of this study will be published in a medical journal but your child will not be identified by name in any publication or presentation of results. However your child's medical notes may be reviewed by the people associated with the study, without your additional permission, should you decide to allow your child to participate in the study.

If you have any further questions, please contact Dr Dulari Gupta or Dr Indira Agarwal (Tel: 0416-2283348 or email dularigupta@gmail.com)

PATIENT CONSENT FORM

Study Title: To study the cardiovascular consequences of Chronic Kidney Disease in children.

Participant's Name:

Hospital Number:

I _____
Father/ Mother/ Guardian of _____declare that:
(please tick the boxes)

I have read the Patient Information Sheet provided to me regarding the study and have clarified any doubts that I had. ( )

I also understand that my child's participation in this study is entirely voluntary and that I am free to withdraw permission to continue to participate at any time without affecting my child's usual treatment or my legal rights. ( )

I understand that my child will not be charged for the tests done under the study but will have to pay for the tests done for standard care. ( )

I understand that the study staff and the institutional ethics committee members will not need my permission to look at my child's records even if I withdraw from the study. I agree to this access. ( )

I understand that my child's identity will not be revealed in any information released to third parties or publishers. ( )

I voluntarily agree to let my child take part in this study. ( )

Name:	**Name of Witness:**
Relation to participant:	**Relation to participant:**
Signature:	**Signature:**
Date:	**Date:**

जानने-समझने के बाद सहमति (फार्म)
मरीजों के लिए जानकारी

बाल-नेफ्रॉलोजी यूनिट
क्रिस्चियन मेडिकल कॉलेज एवं अस्पताल, वेल्लोर

शोध का विषयः बच्चों के हृदय पर क्रानिक रीनल फेलियर (Chronic Renal Failure) **के प्रभाव का अध्ययन।**

आपके बच्चे के हृदय पर क्रानिक रीनल फेलियर (Chronic Renal Failure) का क्या असर होता है इस शोध के लिए आपसे अनुमति की प्रार्थना है।

इस अध्ययन की क्या जरूरत है?

दुनिया भर में बच्चों को गुर्दे (Kidney) की पुरानी बीमारी होती है। बीपी (BP), हृदय की बीमारियां, असमान्य लिपिड घटक आदि के कारण उनके हृदय गति रुकने की सम्भावना बढ़ जाती है। **(सी-के-डी) क्रानिक किडनी डिजीज** (CKD) से पीड़ित बच्चों में 40 प्रतिशत मौतें हृदय के रोगों से होती हैं। नियमित रूप से बीपी (BP), यूएसजी-डॉपलर (USG-Doppler), और एको-टेस्ट (Echo Test) आदि की मदद से हम इन बच्चों के हृदय संबंधी रोगों को जल्दी पहचान सकते हैं। इस जानकारी के बाद हम हृदय रोगों से उन्हें बचाने की कोशिश सकते हैं।

क्या इस अध्ययन से आपके बच्चे को कोई हानि हो सकती है?

हम सभी बच्चों पर कई अतिरिक्त टेस्ट करेंगे:

1) एक सरल टेस्ट द्वारा हम 24-घंटे बच्चों की बीपी (BP) पर निगरानी रखेंगे। इससे बच्चों को कोई दर्द या तकलीफ नहीं होगी।

2) एको-टेस्ट (Echo Test): इस टेस्ट में कोई सुई नहीं चुभोई जाती है। यह दर्द-हीन टेस्ट है। इस टेस्ट से हम हृदय-रोगों की सम्भावना को पहले ही पता कर पायेंगे।

3) यूएसजी-डॉपलर टेस्ट (USG-Doppler Test): इस टेस्ट द्वारा हम गर्दन की नसों की मोटाई जांचकर उनमें जमा वसा की जानकारी हासिल करते हैं।

इन सभी टेस्टों में कोई सुई नहीं चुभोई जाती और न ही कोई रेडियेशन दिया जाता है। इनमें बच्चे को कोई दर्द नहीं होता है। **(सी-के-डी) क्रानिक किडनी डिजीज** (CKD) रोग से ग्रस्त सभी बच्चों को यह तीनों टेस्ट करवाने की सलाह दी जाती है। अभी इन टेस्टों को सामान्य रूप से नहीं किया जाता है।

अगर आपका बच्चा इसमें हिस्सा लेता है, तो उसके साथ क्या किया जाएगा?

अगर आप इस अध्ययन में अपने बच्चे को भाग लेने की अनुमति देते हैं तो हम बच्चे की बीमारियों को एक फार्म पर दर्ज करेंगे। सामान्य खून की जांच के साथ-साथ हम तीन नए टेस्ट करेंगे - 24 घंटे बच्चे की बीपी (BP) पर निगरानी रखेंगे, एको-टेस्ट (Echo Test) और यूएसजी-डॉपलर टेस्ट (USG-Doppler Test) द्वारा सम्भावित हृदय रोगों का पता लगायेंगे। अस्पताल में आपके बच्चे को जो सामान्य उपचार मिल रहा है वो लगातार जारी रहेगा।

अध्ययन शुरू होने के दौरान क्या आप अपने बच्चे को उसमें से निकाल सकते हैं?

बच्चे का इस अध्ययन में शामिल होना पूरी तरह से आपकी मर्जी पर निर्भर करेगा। आप अगर चाहें तो अध्ययन के दौरान कभी भी, किसी भी स्टेज पर आप अपने बच्चे को उसमें से निकाल सकते हैं। अगर आपने बच्चे को निकाला, तो भी अस्पताल में उसका सामान्य उपचार पहले की तरह ही जारी रहेगा।

क्या आपको इन अतिरिक्त टेस्टों के लिए कुछ और पैसे देने होंगे?

ऊपर के तीनों टेस्ट - 24 घंटे बच्चे के बीपी (BP) की निगरानी, एको-टेस्ट (Echo Test) और यूएसजी-डॉपलर टेस्ट (USG-Doppler Test) द्वारा सम्भावित हृदय रोगों का पता लगाने का काम हम **मुफ्त** में करेंगे। अस्पताल में आपके बच्चे का जो सामान्य इलाज चल रहा है वो वैसे ही जारी रहेगा और उसका जो पेमेंट होगा वो आपको देना होगा।

क्या आपकी व्यक्तिगत जानकारी को गोपनीय रखा जाएगा?

इस अध्ययन के नतीजों को किसी मेडिकल जर्नल में छापा जायेगा परन्तु उनमें आपके बच्चे का नाम कहीं पर भी नहीं आयेगा। यह संभव है कि इस अध्ययन के बाद अन्य शोधकर्ता आपके बच्चे के मेडिकल नोट्स की आगे जांच-पड़ताल करें। अगर आप इस अध्ययन के लिए अपनी मंजूरी देते हैं तो इस बाद के शोध के लिए आपकी अनुमति नहीं ली जायेगी।

अगर इस बारे में आप कोई भी सवाल पूछना चाहते हों तो कृपा कर डा दुलारी गुप्ता या डा इंद्रा अग्रवाल से संपर्क करें (टेलीफोन 0416-2283348 **या ईमेलः** dularigupta@gmail.com)

मरीज का सहमति फार्म

शोध का विषयः बच्चों के हृदय पर (सी-के-डी) क्रानिक किडनी डिजीज (CKD) के प्रभाव का अध्ययन करना।

मरीज का नामः .....

अस्पताल का नम्बरः .....

मैं .....
..... का पिता / मां यह घोषित करता/करती हूं कि:

मैंने मरीजों की जानकारी वाले फार्म को अच्छी तरह पढ़ने के बाद अपने सभी प्रश्नों का शंका समाधान कर लिया है। ( )

मुझे यह भी अच्छी तरह पता है कि इस अध्ययन में मेरे बच्चे की भागीदारी पूरी तरह स्वैच्छिक (voluntary) है। अध्ययन के दौरान मैं जब चाहूं अपने बच्चे को इस शोध में से निकाल सकता / सकती हूं। इससे अस्पताल में बच्चे का इलाज सामान्य रूप से चलता रहेगा और उसके कानूनी हकों का कोई उल्लंघन नहीं होगा। ( )

मुझे यह भी पता है कि बच्चे पर जो अतिरिक्त टेस्ट किए जायेंगे उन पर मुझे कोई खर्चा नहीं करना होगा। हां, अस्पताल में इलाज का जो सामान्य खर्च आयेगा वो मुझे देना होगा। ( )

मुझे यह भी पता है कि अगर मैं इस अध्ययन से बच्चे को अलग भी करता / करती हूं तो भी संस्था की नैतिक कमेटी (Ethics Committee) के मेम्बरों को बच्चे की बीमारी के रिकार्ड्स देखने की छूट होगी। मैं इसकी अनुमति देता / देती हूं। ( )

मैं यह भी जानता / जानती हूं कि किसी भी स्टेज पर मेरे बच्चे की पहचान को न तो कहीं छापा जायेगा और न ही किसी तीसरी पार्टी को बताया जायेगा। ( )

मैं स्वेच्छा से अपने बच्चे को इस शोध में शामिल होने की अनुमति देता / देती हूं। ( )

नामः **गवाह का नामः**

बच्चे से रिश्ताः **बच्चे से रिश्ताः**

हस्ताक्षरः **हस्ताक्षरः**

तारीखः **तारीखः**

குழந்தைச் சிறுநீரக பிரிவு

பங்கேற்போருக்குத் தேவையான தகவல்

ஆராய்ச்சியின் தேவை:

முதிர்ந்த சிறுநீரக செயலிழப்பு உலகில் அநேக குழந்தைகளை பாதித்துள்ளது. இவ்வாறான குழந்தைகளுக்கு இரத்த கொதிப்பு, இருதய செயல் இழப்பு, அதிகளவான இரத்த கொழுப்பு சத்து போன்ற பிரச்சனைகள் உண்டாகலாம். இவ்வனைத்தும் இக்குழந்தைகளின் இருதயத்திற்கு பெரிய பாதிப்பை உண்டாக்குகின்றன. இக்குழந்தைகளில் 40% இறப்பின் காரணம் இருதய செயலிழப்பாகும். இக்குழந்தைகளின் இரத்த கொதிப்பு அளவு, கரோடிட் இரத்த குழாயின் இரத்த ஓட்டம மற்றும் இருதய ஸ்கேன் செய்வதின் மூலம் இருதய கோளாறை முன்பே அறியலாம், மற்றும் இதன்னால் ஏற்படும் செயலிழப்பு, இறப்பைத் தவிர்க்கலாம்.

இந்த ஆராய்ச்சியில் பங்கேற்பதால் உங்கள் குழந்தைக்கு தீங்கு விளையுமா?

நாங்கள் சில கூடுதலான பரிசோதனைகள் செய்ய இருக்கிறோம்.

1. 24 மணிநேர இரத்த கொதிப்பளவு கண்காணிக்கப்படும்: இது மிகவும் எளிதான முறை. இதனால் குழந்தைக்கு வலியோ வேறெந்த பிரச்சனைகளோ உண்டாகாது.

2. இருதய ஸ்கேன்: இது குழந்தைகளின் இருதய செயல்பாட்டை அறிய உதவும்.

3. கரோடிட் இரத்தகுளாயின் இரத்த ஓட்டத்தை அறியும் ஸ்கேன்.

இவ்வனைத்தும் குழந்தைகளுக்கு எந்த ஒரு தீங்கும் விளையாது.

உங்கள் குழந்தை பங்கேற்றால் என்ன செய்யப்படும்?

உங்கள் குழந்தையை இவ்வாய்வில் பங்கேற்க அனுமதித்தால், குழந்தை பற்றிய குறிப்புகள் ஒரு வடிவத்தில் குறிக்கப்படும். பொதுவாக செய்யப்படும் பரிசோதனைகளுடன் சேர்த்து இம்மூன்றும் செய்யப்படும். தங்கள் குழந்தைக்கு அளிக்கப்படும் மற்ற சிகிச்சைகள் எந்த பாதிப்புமின்றி தொடரப்படும்.

உங்கள் குழந்தை இவ்வாய்வில் இருந்து பங்கேற்க விருப்பம் இல்லாமல் விலகலாமா?

இந்த ஆய்வில் உங்கள் முழு சம்பந்தத்துடனேயே உங்கள் குழந்தை பங்கேற்கும். நீங்கள் எந்த நேரத்திலும் இந்த ஆய்வில் பங்கேர்க்க விருப்பம் இல்லை என்று சொல்லலாம்.

இதில் செய்யப்படும் பரிசோதனைகளுக்கு பணம் செழுத்த வேண்டுமா?

இவ்வனைத்தும் இலவசமாகவே செய்யப்படும். உங்கள் குழந்தையின் மற்ற பரிசோதனைகள் மற்றும் சிகிச்சை முன்பு போலவே தொடரும்.

உங்கள் குழந்தையின் விவரங்கள் இரகசியமாக வைக்கப்படுமா?

இந்த ஆராய்ச்சியின் முடிவுகள் மருத்துவநூல்களில் பிரசுரிக்கப்படும். ஆனால் உங்கள் குழந்தையின் அடையாளம் எவருக்கும் தெரிவிக்கப்பட மாட்டாது. உங்கள் குழந்தையின் மருத்துவ அறிக்கைகள் உங்கள் சம்மதம் இன்றியும் மற்ற மருத்துவர்களால் பரிசீலிக்கப்படலாம்.

மேலும் விவரங்கள் மற்றும் கேள்விகளுக்கு அணுக வேண்டிய நபர்கள்:

டாக்டர். துலாரி குப்தா

டாக்டர். இந்திரா அகர்வால்

0416 – 2283348 (or) dularigupta@gmail.com

மருத்துவ ஆராய்ச்சியில் பங்கேற்க ஒப்புதல்

ஆராய்ச்சியின் பெயர்:

முதிர்ந்த சிறுநீரக செயலழிப்பினால் பாதிக்கப்பட்ட குழந்தைகளுக்கு உண்டாகும் இருதயத்தின் பாதிப்புகளை ஆராயுதல்.

பங்கேற்போர் பெயர்:

ஆராய்ச்சி எண்:

மருத்துவமனை எண்:

பிறந்த தேதி / வயது (வருடங்களில்):

நான், _____ குழந்தையின் தந்தை / தாய் / பாதுகாப்பாளர் பின்வருமாறு கூறுகின்றேன்.

(பெட்டியில் ✓ குறி இடவும்)

- நான் எனக்கு அளிக்கப்பட்ட தகவல் காகிதத்தை படித்தும் மற்ற சந்தேகங்களை கேட்டும் அறிந்து கொண்டேன். ☐
- நான் என் சொந்த விருப்பத்தின் பேரிலேயே செய்யப்படும் ஆராய்ச்சி என்பதையும், எந்நிலையிலும் நான் இந்த ஆராய்ச்சியிலிருந்து என் குழந்தையின் மருத்துவத்தில் தடைபடாமல் விடுபடலாம் என்பதை அறிவேன். இதனால் என்னுடைய சட்ட சலுகைகளையும் இழக்க மாட்டேன் என்பதையும் அறிவேன். ☐
- இந்த ஆராய்ச்சியின் போது என் குழந்தையின் ஆராய்ச்சி முடிவுகளை நானோ, என் குழந்தையை கவனிக்கும் மருத்துவர்களோ அறிந்து கொள்ள முடியாது என்பதை அறிவேன். ☐
- இந்த ஆராய்ச்சிக்காக நான் பணம் செலுத்தவேண்டியதில்லை என்பதையும், மற்ற வழக்கமான மருத்துவத்திற்கு ஆகும் செலவுகளை நான் ஏற்க வேண்டும் என்பதை அறிவேன். ☐
- என் குழந்தையின் பெயர் மற்றும் அடையாளம் எந்த ஒரு மூன்றாம் நபருக்கோ வெளியிடப்படமாட்டாது என அறிவேன். ☐

நான் என் முழு மனதுடன் என் சொந்த இஷ்டத்தின் பேரில் இந்த ஆராய்ச்சியில் என் குழந்தையை அனுமதிக்கிறேன்.

பெயர்:	சாட்சி பெயர்:
பண்கேர்போரின் உறவு:	பண்கேர்போரின் உறவுமுறை:
கையொப்பம்:	கையொப்பம்:
தேதி:	தேதி:

ANNEXURE IV

LEFT VENTRICULAR MASS TABLES

Age-Specific Reference Intervals for Indexed Left Ventricular Mass in Children

Philip R. Khoury, MS, Mark Mitsnefes, MD, MS, Stephen R. Daniels, MD, PhD, and Thomas R. Kimball, MD, FASE, *Cincinnati, Ohio; and Denver, Colorado*

				Percentile							
Age	Gender	n	Variable	10th	25th	50th	75th	90th	95th	Minimum	Maximum
< 6 mo	Boys	62	LVM	7.22	9.04	10.94	14.16	16.28	17.6	6.27	21.18
			LVMI	40.19	46.92	56.44	66.41	75.72	80.1	32.41	83
	Girls	43	LVM	7.59	9.27	11.15	13.76	16.05	16.5	5.49	28.74
			LVMI	39.05	48.62	55.38	65.98	73.47	85.6	21.22	109.2
6 mo ≤ 2 y	Boys	73	LVM	16.95	20.25	23.88	27.84	32.47	33.7	9.43	36.32
			LVMI	36.17	40.66	44.95	53.29	61.27	68.6	26.71	74.75
	Girls	53	LVM	15.39	17.45	22.25	26.46	31.98	34.6	12.22	35.98
			LVMI	32.91	38.67	42.04	49.85	52.86	57.1	24.18	61.06
2 ≤ 4 y	Boys	124	LVM	24.37	28.52	33.31	38.79	45.48	48.4	13.27	58.13
			LVMI	28.44	33.88	39.5	45.19	48.74	52.4	21.25	77.07
	Girls	84	LVM	24.7	28.4	33.34	38.15	43.88	46.1	17.9	50.98
			LVMI	28.87	31.85	37.88	43.11	47.65	55.3	20.63	66.58
4 ≤ 6 y	Boys	133	LVM	34.36	39.13	45.49	52.62	59.26	63.2	22.92	83.51
			LVMI	27.68	30.68	36.96	40.2	45.12	48.1	18.76	57.25
	Girls	111	LVM	29.24	34.57	39.67	46.59	50.38	57.3	17.68	76.64
			LVMI	25.85	28.06	32.29	36.43	43.47	44.3	18.17	59.25
6 ≤ 8 y	Boys	117	LVM	40.23	45.14	51.73	62.06	70.48	77.4	25.95	97.29
			LVMI	24.47	28.56	31.79	36.28	40.18	44.6	20.27	59.47
	Girls	110	LVM	36.88	40.6	48.38	55.84	65.54	72.1	25.29	89.3
			LVMI	23.15	25.77	29.71	33.15	37.73	43.5	20.11	54.76
8 ≤ 10 y	Boys	111	LVM	45.32	51.49	62.09	73.42	84.61	91.1	32.35	122
			LVMI	22.45	24.85	29.11	34.57	38.25	41	15.24	53.19
	Girls	99	LVM	39.22	48.08	54.76	70.87	75.49	83.6	31.6	91.82
			LVMI	19.07	22.12	26.63	30.37	34.3	36	13.46	44.35
10 ≤ 12 y	Boys	122	LVM	57.76	66.28	74.1	89.43	105.3	111	37.94	124.7
			LVMI	21.88	24.71	28.18	31.87	36.42	38.2	14.72	43.05
	Girls	92	LVM	57.12	62.94	71.66	85.44	98	102	26.53	149.1
			LVMI	20.22	23.25	26.11	29.63	33.05	35.7	13.06	44.88
12 ≤ 14 y	Boys	180	LVM	66.88	82.5	97.76	117.8	138.1	150	51.18	202.3
			LVMI	21.02	24.38	28.8	32.84	39.08	41.4	12.61	47.75
	Girls	144	LVM	60.79	78.37	92.36	108.8	119.8	128	37.56	165.9
			LVMI	20.47	23.63	26.68	29.86	34.65	38.2	10.21	43.59
14 ≤ 16 y	Boys	194	LVM	90.53	106.9	125.7	145.3	167.2	181	38.51	212
			LVMI	22.22	25.11	28.77	33.49	38.47	40.5	8.905	46.01
	Girls	167	LVM	72.67	84.97	98.73	114.7	130	143	39.53	235
			LVMI	20.69	23.55	26.51	29.97	34.89	36.9	12.31	54.33
≥16 y	Boys	151	LVM	93.1	111.3	131.5	154	183.1	204	64.74	256.7
			LVMI	20.72	24.62	29	32.81	37.73	39.4	13.86	46.33
	Girls	103	LVM	73.9	85.06	101.6	118.8	139.5	154	45.48	201.4
			LVMI	20.06	22.94	26.35	31.4	37.93	40	11.21	50.74

LVMI, LVM index.

ANNEXURE V

ABPM TABLES

Ambulatory Blood Pressure Monitoring in Children and Adolescents: Recommendations for Standard Assessment

A Scientific Statement From the American Heart Association Atherosclerosis, Hypertension, and Obesity in Youth Committee of the Council on Cardiovascular Disease in the Young and the Council for High Blood Pressure Research

Elaine Urbina, MD, Chair; Bruce Alpert, MD, FAHA; Joseph Flynn, MD, MS; Laura Hayman, RN, PhD, FAHA; Gregory A. Harshfield, PhD, FAHA; Marc Jacobson, MD, FAHA; Larry Mahoney, MD, FAHA; Brian McCrindle, MD, MPh, FAHA; Michele Mietus-Snyder, MD; Julia Steinberger, MD, MS; Stephen Daniels, MD, PhD, FAHA

Table 3. 90th and 95th Percentiles of Mean Daytime and Nighttime Ambulatory Systolic and Diastolic BP, Stratified According to Gender and Height

	Systolic BP, mm Hg				Diastolic BP, mm Hg			
	Day		Night		Day		Night	
Height, cm	90th pct	95th pct	90th pct	95th pct	90th pct	95th pct	90th pct	95th pct
Boys								
120	120.6	123.5	103.7	106.4	79.1	81.2	61.9	64.1
125	121.0	124.0	104.9	107.8	79.8	81.3	62.2	64.3
130	121.6	124.6	106.3	109.5	79.3	81.4	62.4	64.5
135	122.2	125.2	107.7	111.3	79.3	81.3	62.7	64.8
140	123.0	126.0	109.3	113.1	79.2	81.2	62.9	65.0
145	124.0	127.0	110.7	114.7	79.1	81.1	63.1	65.2
150	125.4	128.5	111.9	115.9	79.1	81.0	63.3	65.4
155	127.2	130.2	113.1	117.0	79.2	81.1	63.4	65.6
160	122.2	132.3	114.3	118.0	79.3	81.3	63.6	65.7
165	131.3	134.5	115.5	119.1	79.7	81.7	63.7	65.8
170	133.5	136.7	116.8	120.2	80.1	82.2	63.8	65.9
175	135.6	138.8	119.1	121.2	80.6	82.8	63.8	65.9
180	137.7	140.9	119.2	122.1	81.1	83.4	63.8	65.8
185	139.8	143.0	120.3	123.0	81.7	84.1	63.8	65.8
Girls								
120	118.5	121.1	105.7	109.0	79.7	81.8	64.0	66.4
125	119.5	122.1	106.4	109.8	79.7	81.8	63.8	66.2
130	120.4	123.1	107.2	110.6	79.7	81.8	63.3	66.0
135	121.4	124.1	107.9	111.3	79.7	81.8	63.4	65.8
140	122.3	125.1	108.4	111.9	79.8	81.8	63.2	65.7
145	123.4	126.3	109.1	112.5	79.8	81.9	63.0	65.6
150	124.6	127.5	109.9	113.1	79.9	81.9	63.0	65.5
155	125.7	128.5	110.6	113.8	79.9	81.9	62.9	65.5
160	126.6	129.3	111.1	114.0	79.9	81.9	92.8	65.4
165	127.2	129.8	111.2	114.0	79.9	81.9	62.7	65.2
170	127.5	130.0	111.2	114.0	79.9	81.8	62.5	65.0
175	127.6	129.9	111.2	114.0	79.8	81.7	62.3	64.7

BP indicates blood pressure; pct, percentile.

Adapted from Wühl et al,[75] with permission from Lippincott Williams & Wilkins.

Ambulatory Blood Pressure Values for Healthy White Children. Adapted from Wühl et al,[75] with permission from Lippincott Williams & Wilkins.

Appendix A. Normal Values for Ambulatory Blood Pressure (mm Hg) for Boys by Height

	Height (cm)													
BP Percentile	120	125	130	135	140	145	150	155	160	165	170	175	180	185
24-hour SBP														
50th	104.5	105.3	106.2	107.2	108.3	109.5	110.9	112.5	114.2	116.1	118.0	119.7	121.5	123.2
75th	109.2	110.1	111.1	112.1	113.3	114.6	116.1	117.7	119.5	121.4	123.2	125.0	126.6	128.2
90th	113.8	114.8	115.9	116.9	118.2	119.5	121.0	122.6	124.4	126.3	128.1	129.8	131.3	132.8
95th	116.8	117.8	118.9	120.0	121.2	122.5	124.0	125.7	127.4	129.3	131.1	132.6	134.1	135.5
99th	122.9	123.9	125.0	126.1	127.3	128.6	130.1	131.7	133.4	135.2	136.8	138.2	139.4	140.5
Daytime SBP														
50th	110.8	111.1	111.5	112.0	112.7	113.7	115.1	116.8	118.6	120.6	122.6	124.4	126.2	128.0
75th	116.2	116.5	116.9	117.4	118.0	119.0	120.4	122.1	124.2	126.4	128.4	130.3	132.2	134.1
90th	121.7	121.9	122.2	122.5	123.0	123.9	125.3	127.1	129.4	131.9	134.1	136.1	138.0	139.9
95th	125.2	125.3	125.5	125.7	126.0	126.9	128.3	130.2	132.7	135.3	137.6	139.6	141.6	143.5
99th	132.6	132.4	132.2	132.0	132.1	132.8	134.2	136.3	139.1	142.2	144.7	146.8	148.6	150.5
Nighttime SBP														
50th	93.6	94.6	95.6	96.7	97.9	99.0	100.1	101.3	102.6	104.1	105.6	107.2	108.7	110.2
75th	98.6	99.8	101.0	102.3	103.6	104.7	105.9	107.1	108.4	109.9	111.5	113.1	114.6	116.1
90th	103.3	104.8	106.3	107.8	109.3	110.6	111.8	113.0	114.3	115.7	117.2	118.8	120.3	121.8
95th	106.3	107.9	109.7	111.4	113.0	114.4	115.7	116.8	118.1	119.4	120.9	122.4	123.9	125.3
99th	112.1	114.2	116.5	118.7	120.8	122.5	123.8	124.9	126.0	127.1	128.4	129.6	131.0	132.2
24-hour DBP														
50th	65.6	65.9	66.1	66.4	66.6	66.9	67.1	67.2	67.3	67.5	67.6	67.8	68.0	68.2
75th	69.7	69.9	70.2	70.4	70.6	70.8	71.0	71.1	71.2	71.3	71.5	71.7	71.8	71.9
90th	73.9	74.1	74.2	74.4	74.5	74.7	74.8	74.8	74.9	75.1	75.3	75.4	75.5	75.6
95th	76.7	76.8	76.9	76.9	77.0	77.1	77.1	77.2	77.3	77.5	77.7	77.8	77.9	78.0
99th	82.7	82.5	82.3	82.1	81.9	81.8	81.8	81.8	81.9	82.2	82.5	82.7	82.9	83.0
Daytime DBP														
50th	72.3	72.3	72.2	72.1	72.1	72.1	72.1	72.1	72.2	72.3	72.6	72.8	73.1	73.4
75th	76.5	76.4	76.3	76.2	76.0	76.0	75.9	75.9	76.0	76.2	76.5	76.8	77.2	77.5
90th	80.2	80.1	79.9	79.7	79.5	79.4	79.3	79.3	79.4	79.7	80.0	80.5	80.9	81.3
95th	82.4	82.2	82.0	81.8	81.5	81.4	81.2	81.2	81.3	81.7	82.1	82.6	83.1	83.6
99th	86.5	86.2	85.9	85.6	85.2	85.0	84.8	84.8	85.0	85.4	86.0	86.6	87.3	87.9
Nighttime DBP														
50th	54.3	54.8	55.1	55.5	55.8	56.0	56.2	56.2	56.3	56.5	56.7	56.9	57.1	57.3
75th	57.6	58.2	58.8	59.2	59.6	59.9	60.1	60.2	60.2	60.3	60.5	60.6	60.8	60.9
90th	60.7	61.4	62.1	62.7	63.2	63.5	63.7	63.8	63.8	63.9	63.9	64.0	64.1	64.2
95th	62.6	63.4	64.2	64.8	65.4	65.8	66.0	66.0	66.0	66.0	66.1	66.1	66.1	66.2
99th	66.2	67.2	68.2	69.0	69.7	70.1	70.4	70.4	70.3	70.3	70.2	70.1	70.0	69.9
24-hour MAP														
50th	77.5	78.1	78.7	79.3	79.9	80.5	81.1	81.7	82.3	83.1	83.9	84.7	85.5	86.3
75th	81.8	82.4	83.0	83.5	84.1	84.6	85.2	85.9	86.6	87.3	88.1	89.0	89.8	90.7
90th	86.3	86.7	87.2	87.6	88.0	88.5	89.1	89.7	90.3	91.1	91.9	92.7	93.5	94.3
95th	89.3	89.6	89.9	90.2	90.5	90.9	91.4	91.9	92.6	93.3	94.0	94.8	95.6	96.4
99th	95.9	95.7	95.5	95.4	95.4	95.6	95.9	96.3	96.7	97.4	98.0	98.7	99.4	100.1
Daytime MAP														
50th	83.8	84.1	84.3	84.5	84.7	85.0	85.4	85.8	86.4	87.1	88.0	89.0	90.0	91.0
75th	88.5	88.7	88.9	89.0	89.1	89.4	89.6	90.1	90.7	91.6	92.6	93.7	94.9	96.1
90th	92.9	93.0	93.1	93.1	93.1	93.2	93.4	93.8	94.5	95.4	96.5	97.7	99.0	100.3
95th	95.6	95.6	95.6	95.5	95.5	95.5	95.7	96.0	96.7	97.7	98.8	100.1	101.4	102.8
99th	101.0	100.7	100.5	100.2	99.9	99.7	99.8	100.1	100.8	101.7	102.9	104.3	105.7	107.1
Nighttime MAP														
50th	67.6	68.3	69.0	69.6	70.1	70.6	71.2	71.9	72.7	73.6	74.5	75.4	76.2	
75th	71.9	72.7	73.4	73.9	74.4	74.9	75.4	76.0	76.8	77.6	78.3	79.1	79.8	
90th	76.6	77.3	77.9	78.3	78.6	78.9	79.2	79.7	80.3	80.9	81.5	82.1	82.7	
95th	80.0	80.5	80.9	81.2	81.3	81.4	81.5	81.9	82.3	82.8	83.3	83.8	84.3	
99th	88.1	87.8	87.6	87.2	86.7	86.3	86.0	86.0	86.1	86.3	86.5	86.8	87.0	

BP indicates blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; and MAP, mean arterial pressure.

Appendix B. Normal Values for Ambulatory Blood Pressure (mm Hg) for Girls by Height

	Height (cm)											
BP Percentile	120	125	130	135	140	145	150	155	160	165	170	175
24-hour SBP												
50th	104.0	105.0	106.0	106.8	107.6	108.7	109.9	111.2	112.4	113.7	115.0	116.4
75th	108.2	109.3	110.3	111.2	112.1	113.2	114.6	115.9	117.0	118.0	119.2	120.4
90th	112.0	113.2	114.3	115.3	116.2	117.4	118.7	120.0	121.0	121.8	122.8	123.8
95th	114.3	115.6	116.7	117.7	118.7	119.9	121.2	122.5	123.3	124.1	124.9	125.8
99th	118.8	120.1	121.3	122.4	123.4	124.6	126.0	127.1	127.7	128.2	128.8	129.3
Daytime SBP												
50th	110.0	110.5	111.0	111.6	112.2	113.1	114.3	115.6	117.0	118.3	119.8	121.2
75th	114.4	115.0	115.7	116.3	117.0	118.1	119.4	120.7	121.9	123.1	124.2	125.3
90th	118.2	119.0	119.7	120.4	121.3	122.5	123.9	125.2	126.4	127.3	128.1	128.9
95th	120.4	121.3	122.1	122.9	123.8	125.1	126.5	127.9	129.1	129.8	130.5	131.0
99th	124.5	125.5	126.4	127.4	128.5	129.9	131.5	133.0	134.0	134.5	134.8	135.0
Nighttime SBP												
50th	95.0	95.7	96.4	96.9	97.5	98.1	98.9	100.0	101.1	102.2	103.4	104.6
75th	99.4	100.3	101.2	101.9	102.6	103.4	104.4	105.5	106.4	107.3	108.2	109.2
90th	103.3	104.4	105.5	106.5	107.5	108.5	109.5	110.5	111.2	111.8	112.4	113.1
95th	105.6	106.9	108.1	109.3	110.4	111.6	112.7	113.6	114.1	114.4	114.8	115.3
99th	109.8	111.5	113.1	114.7	116.2	117.7	118.9	119.5	119.6	119.4	119.3	119.4
24-hour DBP												
50th	65.9	65.9	66.0	66.1	66.2	66.3	66.5	66.7	67.0	67.4	68.0	68.6
75th	68.6	68.9	69.2	69.5	69.8	70.1	70.4	70.6	70.7	71.0	71.3	71.6
90th	70.9	71.4	71.9	72.4	72.9	73.4	73.8	74.0	74.1	74.2	74.4	74.5
95th	72.2	72.8	73.4	74.1	74.7	75.3	75.7	76.0	76.1	76.2	76.2	76.2
99th	74.6	75.3	76.2	77.1	77.9	78.7	79.3	79.7	79.9	79.9	79.9	79.7
Daytime DBP												
50th	73.2	72.8	72.4	72.1	71.8	71.7	71.8	72.0	72.4	73.1	73.9	74.8
75th	76.9	76.6	76.4	76.2	76.1	76.1	76.1	76.2	76.4	76.8	77.3	77.8
90th	80.1	79.9	79.8	79.8	79.7	79.8	79.9	79.9	79.9	80.0	80.2	80.5
95th	81.9	81.8	81.8	81.8	81.9	82.0	82.0	82.0	82.0	81.9	82.0	82.0
99th	85.3	85.3	85.4	85.6	85.8	85.9	86.0	85.9	85.7	85.4	85.2	84.9
Nighttime DBP												
50th	55.4	55.3	55.1	54.8	54.6	54.4	54.3	54.4	54.6	54.9	55.1	55.4
75th	59.5	59.5	59.4	59.3	59.1	58.9	58.8	58.7	58.8	58.9	61.0	59.3
90th	63.1	63.3	63.4	63.4	63.3	63.1	63.0	62.9	62.9	62.9	66.9	63.1
95th	65.2	65.5	65.7	65.8	65.8	65.7	65.6	65.5	65.5	65.5	70.8	65.5
99th	69.1	69.6	70.1	70.4	70.6	70.8	70.8	70.7	70.7	70.6	79.0	70.4
24-hour MAP												
50th	77.2	77.8	78.3	78.7	79.2	79.7	80.2	80.8	81.5	82.3	83.1	84.0
75th	80.6	81.2	81.8	82.4	82.9	83.5	84.1	84.7	85.3	85.9	86.6	87.4
90th	83.6	84.2	84.9	85.5	86.1	86.7	87.3	87.9	88.4	88.9	89.5	90.1
95th	85.3	86.0	86.7	87.4	88.0	88.6	89.2	89.7	90.2	90.6	91.1	91.7
99th	88.5	89.2	89.9	90.6	91.3	91.9	92.5	93.0	93.3	93.6	94.0	94.5
Daytime MAP												
50th	83.3	83.7	84.0	84.1	84.3	84.5	84.9	85.5	86.2	87.0	88.0	88.9
75th	87.4	87.9	88.2	88.5	88.7	88.9	89.3	89.8	90.3	90.9	91.6	92.2
90th	90.9	91.5	91.9	92.2	92.4	92.7	93.0	93.4	93.7	94.1	94.5	94.9
95th	92.9	93.6	94.0	94.4	94.6	94.9	95.1	95.4	95.6	95.8	96.1	96.4
99th	96.6	97.4	97.9	98.3	98.6	98.8	99.0	99.0	99.0	99.0	99.0	99.1
Nighttime MAP												
50th	68.0	68.2	68.4	68.5	68.7	69.0	69.3	69.8	70.4	71.2	72.0	72.8
75th	72.6	72.7	72.9	73.0	73.2	73.5	73.9	74.3	74.8	75.4	76.1	76.9
90th	76.8	76.9	77.0	77.2	77.4	77.7	78.0	78.3	78.6	79.1	79.6	80.3
95th	79.5	79.4	79.6	79.7	79.9	80.2	80.4	80.6	80.8	81.2	81.6	82.2
99th	84.6	84.4	84.5	84.6	84.8	85.0	85.0	85.0	85.0	85.0	85.3	85.6

BP indicates blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; and MAP, mean arterial pressure.

ANNEXURE VI

ABPM DATA SAMPLE

Patient Information

Name: DWIVEDI.SUDITI
ID: 379943D
Date-of-Birth: 01/01/1900 00:00 Mon
Age: 112 Years
Medications: Dose: Time:

Gender: Female
Height:
Weight:
Race: Unspecified
Physician: Agarwal,Dr Indira
Nurse/Technician:
Duration: 24:40
Scan Start: 24/11/2012 11:48 Sat
Scan End: 25/11/2012 12:28 Sun
Successful Reading(s): 58 98%
Indications:

Overall Summary

	AVG	STD		MIN	MAX	Dipping
Systolic:	133	6.59	mmHg	114 (05:08 Sun)	155 (20:08 Sat)	4.5%
Diastolic:	86	5.89	mmHg	73 (16:28 Sat)	100 (12:48 Sat)	1.2%
MAP:	99	5.91	mmHg	89	115	2.0%
Pulse Pressure:	47	5.52	mmHg	36	67	
Heart Rate:	102	4.64	bpm	93	115	

	Reading(s)	Time
Percent of Systolic above limits:	39.7%	48.6%
Percent of Diastolic above limits:	67.2%	72.9%

Wake Period(s) 06:00 - 22:00

	AVG	STD		MIN	MAX
Systolic:	134	6.29	mmHg	122 (18:48 Sat)	155 (20:08 Sat)
Diastolic:	86	6.06	mmHg	73 (16:28 Sat)	100 (12:48 Sat)
MAP:	100	5.99	mmHg	89	115
Pulse Pressure:	47	5.57	mmHg	36	67
Heart Rate:	102	4.53	bpm	94	115

	Reading(s)	Time
Percent of Systolic readings > 135mmHg:	32.0%	28.7%
Percent of Diastolic readings > 85mmHg:	62.0%	60.7%

Number of Wake Period(s) readings: 50

Sleep Period(s) 22:00 - 06:00

	AVG	STD		MIN	MAX
Systolic:	128	6.63	mmHg	114 (05:08 Sun)	135 (23:08 Sat)
Diastolic:	85	4.98	mmHg	77 (05:08 Sun)	92 (22:08 Sat)
MAP:	98	5.41	mmHg	89	105
Pulse Pressure:	42	2.72	mmHg	37	45
Heart Rate:	98	4.07	bpm	93	106

	Reading(s)	Time
Percent of Systolic readings > 120mmHg:	87.5%	92.9%
Percent of Diastolic readings > 70mmHg:	100%	100%

Number of Sleep Period(s) readings: 8

Interpretation

Signed **Date**

Facility:
Name: **DWIVEDI,SUDITI**

RAW DATA SUMMARY

ID: 379943D
Hookup:11:48

#		Time	Sys	Dia	MAP	PP	HR	Diary Activity
1	M	11:48 Sat	132	87	102	45	105	
2		12:08	133	87	102	46	102	
3		12:28	132	86	97	46	105	
4		12:48	143	100	112	43	109	
5		13:08	140	95	115	45	113	
6		13:28	139	96	107	43	111	
7		13:48	142	91	107	51	102	
8		14:08	140	83	100	57	98	
9		14:28	131	77	90	54	98	
10		14:48	132	78	92	54	100	
11		15:08	130	77	92	53	97	
12		15:28	125	74	89	51	99	
13		15:48	125	74	89	51	97	
14		16:08	125	74	92	51	107	
15		16:28	126	73	89	53	105	
16		16:48	133	85	95	48	102	
17		17:08	127	81	96	46	104	
19	R	17:31	130	89	98	41	102	
20		17:48	137	81	101	56	99	
21		18:08	133	89	99	44	99	
22		18:28	134	85	97	49	95	
23		18:48	122	80	91	42	97	
24		19:08	134	89	100	45	99	
25		19:28	134	93	105	41	100	
26		19:48	143	88	103	55	104	
27		20:08	155	88	111	67	110	
28		20:28	130	85	98	45	106	
30	R	20:51	134	88	101	46	105	
31		21:08	133	81	98	52	102	
32		21:28	134	90	101	44	104	
33		21:48	133	89	101	44	100	
34		22:08	133	92	103	41	106	
35		23:08	135	90	105	45	101	
37	R	00:11 Sun	128	86	98	42	99	
38		01:08	128	83	95	45	94	
39		02:08	133	89	102	44	93	
40		03:08	126	85	98	41	97	
41		04:08	125	81	93	44	98	
42		05:08	114	77	89	37	99	
43		06:08	129	85	96	44	103	
44		06:28	127	88	99	39	102	
46		07:08	137	87	103	50	108	
47		07:28	135	87	99	48	102	
49	R	07:51	122	86	96	36	107	
50		08:08	131	89	100	42	115	
51		08:28	141	94	106	47	104	
53	R	08:50	140	96	108	44	107	
54		09:08	126	84	92	42	102	
55		09:28	135	86	102	49	100	
56		09:48	133	82	100	51	94	
57		10:08	122	85	95	37	100	
58		10:28	138	90	103	48	98	
59		10:48	135	89	103	46	100	
60		11:08	133	89	103	44	98	
61		11:28	138	86	100	52	97	
62		11:48	138	89	103	49	101	
63		12:08	136	89	101	47	104	
64		12:28	138	95	107	43	100	

R = Auto Retry
M = Manual Initiated

EE = Event Edit
ME = Manual Edit

AE = Auto Edit
< > = Estimated

	Systolic	Diastolic	MAP	PP	HR
	240	150	200	150	200
	70	40	40	20	20

Printed 14.12.2012 03 09 21 Fri
Auto Edit Limit Max
Auto Edit Limit Min

ABSTRACT

TITLE OF THE ABSTRACT : A prospective cross-sectional study of the cardiovascular consequences in children with Chronic Kidney Disease
DEPARTMENT : Department of Paediatrics
NAME OF THE CANDIDATE : Dr Dulari Gupta
DEGREE AND SUBJECT : MD Paediatrics
NAME OF THE GUIDE : Dr Indira Agarwal

OBJECTIVES: Describe the objectives of your study (maximum 30 words)

To study prevalence of cardiovascular risks amongst children with CKD by measuring Left Ventricular Dysfunction using ECHO and prevalence of Hypertension both manifest (clinic BP Readings) and masked (Ambulatory BP monitoring).

METHODS: Explain the clinical and statistical methods used (maximum 100 words)

Children, aged 1-18 years with Chronic Kidney Disease who presented to the Pediatric Nephrology OPD, General Pediatric OPD or Inpatient Facilities were recruited.
Informed consent was taken from all parents. A detailed history and complete physical examination was done. Routine Blood tests (Hemoglobin, Sodium, Potassium, Creatinine, Urea, Calcium, Phosphorus, Alkaline Phosphatase, Bicarbonate, Parathyroid hormone levels) were noted. The mean of 3 clinic BP readings was recorded. Ambulatory BP was performed over a 24 hour period using the Spacelabs Healthcare machine. ECHO was done to determine Left Ventricular Hypertrophy. Data was analyzed using SPSS software and chi square tests of significance.

RESULTS: Summarise the findings and conclusions of your study (maximum 90 words)

Forty six children with CKD were studied (Male: Female 4.1:1). Obstructive uropathy was found to be the commonest cause of CKD. Forty two children had cardiovascular morbidity (93.1%). LVH was present in 28.26 % children. Clinic Hypertension was present in 21.7% .ABPM helped to detect latent hypertension in 32 additional children who had normal clinic BP.

Left Ventricular Hypertrophy was found to linearly correlation with the ABPM and Clinic BP. Univariate analysis showed BP Index and Clinic BP to significantly contribute to LVH.

We conclude that latent Hypertension is common amongst children with CKD. We recommend ABPM in every child with CKD to detect hypertension and monitor adequacy of hypertensive control.

THESIS DATA SHEET

S no	name	id	sex	age	height	weight	bsa	bmi	cause	dura-tion	gfr	class	dia-lysis	Duration of Dialysis	HT	Duration of HT	HT control	no. of anti HT
1	surya	813056b	0	13	139	27.28	1.05	14.1	1	20	18.36	2	1	4	1	4	1	2
2	meghana	028448f	1	6	108	18	0.73	15.4	1	7	18.56	1	0	0	1	5	0	3
3	alok yadav	81506d	0	5	109	16.6	0.71	14	3	24	33.31	0	0	0	1		0	3
4	amar kumar das	105944f	0	13	165	40.3	1.4	14.8	4	48	32.02	0	0	0	1	1	0	1
5	tanvir sheam	678614d	0	15	148.8	31.8	1.17	14.5	1	144	49.6	0	0	0	1	22	1	2
6	kiranjit biswas	785907d	0	12	137	31.1	1.1	16.6	3	108	45.67	0	0	0	1	9	1	2
7	nazmus sakib	841588c	0	12	144	56.7	1.47	27.3	4	84	27.5	1	0	0	0	0	1	1
8	selv vinayagam	976429d	0	14	158	53.7	1.53	21.5	3	6	13.6	2	1	8	1	1	0	0
9	vignesh n	661380d	0	16	159.5	38.6	1.34	15.3	1	23	64.91	2	1	20	1	23	0	3
10	monisha	978513d	1	14	135.5	27.7	1.03	15.2	3	8	12.76	1	0	0	0	0	1	0
11	vishnu	310052d	0	5	109.6	15.8	0.7	13.3	3	41	6.26	2	1	0	1	13	0	2
12	shubham kumar	678186d	0	16	157	37.8	1.31	15.3	4	24	23.94	1	0	0	1	24	1	2
13	keshav ram sahu	030692f	0	9	117	19	0.79	13.9	3	7	7.31	2	1	3	1	3	0	4
14	premnath	979489b	0	11	145.7	41	1.28	19.5	4	126	26.49	1	0	0	1	24	1	2
15	prthibiraj barik	193478f	0	15	166	41.8	1.43	15.2	4	2	10.2	1	1	1	1	1	1	2
16	wahengbam dashibala	203196f	1	14	146	34.4	1.2	16.1	1	24	37.18	0	0	0	0	0	1	0
17	akhas dom	060141f	1	11	123	22.6	0.89	14.9	4	7	15.91	1	0	0	1	7	1	2
18	mohammed s	228209f	0	13	157	58.9	1.59	23.9	1	6	41.47	0	0	0	0	0	1	0
19	rozhan ali	047248c	0	11	108	16.78	0.71	14.3	4	132	37.43	0	0	0	1	3	1	1
20	raja v	539831d	0	12	113	18.56	0.76	14.5	3	33	22.86	2	1	30	0	0	1	0
21	rishi raghavendran	228705f	0	13	150	39.2	1.29	17.4	3	48	31.16	0	0	0	0	0	1	1
22	vijaya priya	608170d	1	14	153	35	1.25	15	3	3	38.42	0	0	0	0	0	1	1
23	anand s	134526c	0	11	134	25.5	0.99	14.2	4	132	8.69	2	1	3	1	4	1	3
24	bitan bera	239720f	0	11	131	23.6	0.94	13.8	3	7	21.48	2	1	3	1	3	0	4
25	parkavi	551548d	1	13	124.5	20.7	0.86	13.5	4	120	22.9	1	0	0	1	24	1	1
26	gangadhara	947648d	0	11	135	25.3	0.99	13.9	4	96	29.26	1	0	0	0	0	0	0
27	sudharson	896410d	0	13	156	45.2	1.41	18.6	4	154	50.79	0	0	0	1	0	0	3
28	debham saha	954703c	0	17	168	46	1.5	16.3	4	60	50.91	0	0	0	1	33	1	3
29	sudipta mondol	259172f	0	11	148	38	1.26	17.3	1	3	23.49	1	1	1	1	2	1	1
30	mohammed zaith	589689c	0	8	119	20.2	0.82	14.3	4	89	19.36	1	0	0	1	12	1	1
31	somojeet modak	260990f	0	9	134	22.8	0.95	12.7	4	114	36.85	0	0	0	0	0	0	1
32	roshan kumar	827840b	0	12	149	33	1.19	14.9	3	14	30.68	0	0	0	0	0	0	0
33	arvind	163511f	0	14	136	29	1.06	15.7	4	150	32.94	0	0	0	0	0	0	1
34	ravindran	152643c	0	11	129.2	23.86	0.94	14.3	3	132	30.9	0	0	0	0	0	1	1
35	furquanul haque	562142c	0	16	143.2	33.1	1.16	16.2	4	120	16.46	1	0	0	0	0	0	1
36	jatin sharma	220366f	0	7	107	13.8	0.65	12.1	0	3	29.72	1	0	0	0	0	0	0
37	soumitra manna	779138b	1	13	134.6	28.8	1.04	16	4	144	21.65	1	0	0	0	0	0	0
38	samuvel	045336c	0	12	126.2	23	0.91	14.5	3	132	29.45	1	0	0	1	48	1	1
39	Karthik	497918B	0	14	146	45.8	1.35	21.5	4	172	23.44	1	0	0	0	0	0	0
40	indumathy	852689d	1	6	110	15.5	0.7	12.8	4	41	32.35	0	0	0	0	0	0	1
41	niveda	132557d	1	14	138.2	30.8	1.1	16.2	3	36	26.26	1	0	0	0	0	0	1
42	adarsh	338748d	0	8	128	33	1.07	20.1	3	96	43.73	0	0	0	1	84	0	2
43	ramana	915292c	0	6	115.4	18	0.77	13.6	4	61	42.17	0	0	0	0	0	0	0
44	muniyappan	669607d	0	3	77	10.8	0.46	18.2	4	32	13.07	2	0	0	0	0	0	1
45	teja semha	450147d	0	7	105	17	0.7	15.4	3	24	25.9	1	0	0	0	0	0	0
46	Suditi dwivedi	379943d	1	13	137	27.4	1.04	14.6	4	24	7.61	2	1	10	1	8	0	3

THESIS DATA SHEET

S no	name	id	acei	arb	Calcium Channel Blockers	Beta Block-ers	ab	vaso-dilators	Ca	rocal-trol	Iron	erythro-poietin	Bicarbo-nate	calcit-riol	clinic BP	clinic BP1	clinic BP2
1	surya	813056b	0	1	1	0	0	0	1	1	0	0	0	0	110	120	92
2	meghana	028448f	1	1	1	0	0	0	0	0	0	0	1	0	122	134	124
3	alok yadav	81506d	1	0	1	1	0	0	1	0	1	0	1	0	115	101	94
4	amar kumar das	105944f	0	0	0	1	0	0	0	1	0	1	1	0	120	120	150
5	tanvir sheam	678614d	1	0	1	0	0	0	1	0	1	0	0	0	132	133	109
6	kiranjit biswas	785907d	1	0	1	0	0	0	1	0	1	0	0	0	82	116	103
7	nazmus sakib	841588c	1	0	0	0	0	0	1	1	1	1	1	1	120	105	128
8	selv vinayagam	976429d	0	0	0	0	0	0	1	1	1	0	1	0	151	142	133
9	vignesh n	661380d	1	0	1	1	0	0	0	1	1	0	0	0	184	167	170
10	monisha	978513d	0	0	0	0	0	0	1	1	1	1	1	0	110	120	108
11	vishnu	310052d	1	0	1	0	0	0	1	0	1	1	1	0	148	147	151
12	shubham kumar	678186d	0	0	1	0	0	1	1	0	1	0	1	0	120	126	148
13	keshav ram sahu	030692f	1	0	1	0	1	1	1	1	1	0	1	0	132	132	148
14	premnath	979489b	1	0	1	0	0	0	0	0	1	0	0	0	104	124	120
15	prthibiraj barik	193478f	0	0	1	0	1	0	1	1	1	0	0	0	114	134	120
16	wahengbam dashibala	203196f	0	0	0	0	0	0	0	0	0	0	0	0	117	144	133
17	akhas dom	060141f	1	0	1	0	0	0	1	1	1	0	1	0	100	123	120
18	mohammed s	228209f	0	0	0	0	0	0	0	0	0	0	0	0	100	134	119
19	rozhan ali	047248c	1	0	0	0	0	0	1	1	1	0	1	0	107	97	96
20	raja v	539831d	0	0	0	0	0	0	1	1	1	1	1	0	115	113	119
21	rishi raghavendran	228705f	1	0	0	0	0	0	0	1	1	0	1	0	98	99	117
22	vijaya priya	608170d	1	0	0	0	0	0	0	1	1	0	0	1	99	120	120
23	anand s	134526c	1	0	1	0	1	0	1	0	1	1	1	0	89	104	124
24	bitan bera	239720f	0	1	1	0	1	1	1	1	1	1	0	0	140	139	126
25	parkavi	551548d	1	0	0	0	0	0	0	1	1	0	1	0	106	116	111
26	gangadhara	947648d	0	0	0	0	0	0	1	1	1	1	1	1	93	114	99
27	sudharson	896410d	0	1	1	0	1	0	1	1	1	1	1	0	113	137	129
28	debham saha	954703c	1	1	1	0	0	0	1	0	1	0	1	0	129	120	121
29	sudipta mondol	259172f	0	0	1	0	0	0	0	0	1	1	1	0	116	114	118
30	mohammed zaith	589689c	1	0	0	0	0	0	1	1	1	0	1	0	81	90	96
31	somojeet modak	260990f	1	0	0	0	0	0	1	0	1	0	1	1	102	100	94
32	roshan kumar	827840b	0	0	0	0	0	0	1	1	1	1	1	0	108	126	119
33	arvind	163511f	1	0	0	0	0	0	1	1	1	0	1	0	100	110	95
34	ravindran	152643c	0	1	0	0	0	0	1	1	1	0	1	0	94	90	98
35	furquanul haque	562142c	1	0	0	0	0	0	1	1	1	1	1	0	88	88	104
36	jatin sharma	220366f	0	0	0	0	0	0	1	1	1	1	0	1	86	96	97
37	soumitra manna	779138b	0	0	0	0	0	0	1	1	1	0	1	0	109	117	113
38	samuvel	045336c	1	0	0	0	0	0	1	0	1	0	1	0	92	100	90
39	Karthik	497918B	0	0	0	0	0	0	1	1	1	0	1	0	97	90	90
40	indumathy	852689d	1	0	0	0	0	0	1	0	1	0	1	0	78	74	106
41	niveda	132557d	1	0	0	0	0	0	1	1	1	0	0	0	90	90	124
42	adarsh	338748d	0	0	1	1	0	0	1	0	1	0	1	0	114	104	114
43	ramana	915292c	0	0	0	0	0	0	1	0	1	0	1	0	110	99	92
44	muniyappan	669607d	1	0	0	0	0	0	1	1	0	0	0	1	80	82	100
45	teja semha	450147d	0	0	0	0	0	0	1	1	1	0	1	0	98	98	94
46	Suditi dwivedi	379943d	0	1	1	1	0	0	1	1	1	1	1	0	113	130	120

THESIS DATA SHEET

S no	name	id	clinic BP3	clinic BP4	clinic BP5	avg SBP	avgDBP	Clinic BP	hb	so-dium	potas-sium	bicar-bonat	calcium	phos-phorus	creat	urea	pth	upuc	CaPO4	lvidd
1	surya	813056b	60	70	58	107.3333333	62.666667	A	8	136	5.5	17	9.3	3.6	5.3	59	609.9		33.48	4.9
2	meghana	028448f	89	84	94	126.6666667	89	P	8.4	138	4.1	23	7.3	3.5	3.2	105	12	8.79	25.55	4.1
3	alok yadav	81506d	59	50	47	103.3333333	52	A	11.7	141	7.2	20	9.6	4.6	1.8		79.6	0.17	44.16	3.3
4	amar kumar das	105944f	89	90	100	130	93	P	12	135	5.1	19	9.5	3.5	3.6	32	457.9		33.25	4.8
5	tanvir sheam	678614d	106	84	81	124.6666667	90.333333	P	13	135	4.8	20	10.1	4.5	2.1		171.7	2.04	45.45	3.8
6	kiranjit biswas	785907d	42	56	70	100.3333333	56	A	10.4	128	5.5	22	9.7	4.7	2.1	148	149.5	4.84	45.59	4.3
7	nazmus sakib	841588c	80	72	78	117.6666667	76.666667	A	11.9	136	4.4	21	8.5	4.2	2.8	100	201.5	7.2	35.7	3.6
8	selv vinayagam	976429d	99	84	83	142	88.666667	P	10.5	129	4	19	8.5	5.7	8.13	168	867.8		48.45	3.9
9	vignesh n	661380d	129	110	116	173.6666667	118.33333	P	11.7	135	4.5	23	9.1	4.6	1.72	35	290.8		41.86	4.2
10	monisha	978513d	60	70	57	112.6666667	62.333333	A	7.2	141	5.6	17	8.3	3.5	5.84	146	630.2		29.05	3.9
11	vishnu	310052d	122	122	115	148.6666667	119.66667	P	7	133	4.6	27	9.9	5.1	9.63	127	170.7		50.49	3.7
12	shubham kumar	678186d	67	85	86	131.3333333	79.333333	A	11	138	3.9	22	8.8	4.3	4.59	64	229		37.84	3.9
13	keshav ram sahu	030692f	92	98	98	137.3333333	96	P	10.2	137	5.4	20	8.7		11.2	159	891.7		0	4.7
14	premnath	979489b	61	90	86	116	79	A	9.6	132	4.5	18	7.6	4	3.85	83	2.5		30.4	4.2
15	prthibiraj barik	193478f	70	78	67	122.6666667	71.666667	A	7	137	5.5	14	8.8	5.7	11.39	114		3.67	50.16	4.8
16	wahengbam dashibala	203196f	87	101	83	131.3333333	90.333333	P	11.6	138	4.2	21	8.8	4.6	2.16	29	177.9	3.96	40.48	3.6
17	akhas dom	060141f	70	93	80	114.3333333	81	A	8.1	135	6.2	21	9.6	5.5	5.41	128	178.2		52.8	2.4
18	mohammed s	228209f	60	65	55	117.6666667	60	A	10.7	137	4.7	38	8.3	4	2.65	29	57.4	0.13	33.2	3.6
19	rozhan ali	047248c	59	56	61	100	58.666667	A	8.1	135	7.1	12	8.8	2.9	2.02	87	537.8		25.52	3.5
20	raja v	539831d	78	75	80	115.6666667	77.666667	A	11.5	142	4.8	16	10	6.1	3.46	53	955.4		61	3.9
21	rishi raghavendran	228705f	75	63	86	104.6666667	74.666667	A	8.9	138	3.5	20	8.4	2.6	3.37	39	1327		21.84	3.4
22	vijaya priya	608170d	67	86	70	113	74.333333	A	8.9	135	4	19	9	3.3	2.19		259.4		29.7	3.5
23	anand s	134526c	41	51	90	105.6666667	60.666667	A	9.9	134	6	22	9.2	5.5	8.98	154	59.1		50.6	3.6
24	bitan bera	239720f	114	99	78	135	97	P	9.4	140	3.6	19	8.9	5	4.27	41	261.7		44.5	4.3
25	parkavi	551548d	72	90	68	111	76.666667	A	11.4	134	5.3	16	9.3	5	2.99	79	200	4.42	46.5	3.7
26	gangadhara	947648d	52	70	70	102	64	A	11.7	135	5.4	19	7.9	3.2	3.23		771.5	2.25	25.28	3.5
27	sudharson	896410d	55	68	90	126.3333333	71	P	11.7	142	4.8	19	8.4	4.7	2.15	29	99.9	0.74	39.48	4.3
28	debham saha	954703c	61	80	64	123.3333333	68.333333	A	12.7	134	4.8	15	9	3.5	2.31		91.5	1.46	31.5	2.9
29	sudipta mondol	259172f	74	52	68	116	64.666667	A	9.7	132	3.3	15	8.2	5.9	4.41	90	29	0.01	48.38	4.3
30	mohammed zaith	589689c	43	50	53	89	48.666667	A	10.6	133	4.9	20	9.1	5.7	3.38	87	85.6	0.97	51.87	3
31	somojeet modak	260990f	77	65	65	98.66666667	69	A	10	129	4.4	18	8.2	3.7	2	67	45		30.34	4.2
32	roshan kumar	827840b	69	80	71	117.6666667	73.333333	A	10.3	136	4.6	18	9.3	6.4	3.4	63	5	1.54	59.52	4
33	arvind	163511f	50	78	54	101.6666667	60.666667	A	10.7	141	4	18	8.2	2.7	2.89	51	466.6		22.14	3.6
34	ravindran	152643c	60	40	50	94	50	A	8.5	139	4.7	21	8.9	5.9	2.3	56	180.3	1.18	52.51	3.9
35	furquanul haque	562142c	41	42	63	93.33333333	48.666667	A	10.1	134	5	12	7.7	5.4	6.09	137	913.1	1.2	41.58	3.5
36	jatin sharma	220366f	52	63	59	93	58	A	7.1	137	3.3	12	7.3	2.3	1.98	75	1713	2.21	16.79	3.1
37	soumitra manna	779138b	67	65	74	113	68.666667	A	11	139	4.2	16	9.1	3.8	3.42	106	114.5	2.49	34.58	3.1
38	samuvel	045336c	46	70	60	94	58.666667	A	9.2	138	4.7	22	8.6	4.5	3		81.9		38.7	3.4
39	Karthik	497918B	48	60	60	92.33333333	56	A	12.6	138	4	18	8.4	4.8	4.36	74	386.3		40.32	3.9
40	indumathy	852689d	48	36	65	86	49.666667	A	9.5	133	4.2	26			1.87	79	164.3		0	2.8
41	niveda	132557d	50	50	74	101.3333333	58	A	6.9	138	5.6	19	8.6	5.5	2.89		440		47.3	4
42	adarsh	338748d	73	79	60	110.6666667	70.666667	A	14.1	141	3.5	18	9	4.2	1.61	41	137.3	0.31	37.8	3.8
43	ramana	915292c	70	67	60	100.3333333	65.666667	A	12.3	139	4.3	14	9.2	6.4	1.5		41.8		58.88	3.3
44	muniyappan	669607d	50	64	70	87.33333333	61.333333	A	9.2	136	6	19	9.5	3.9	3.24		267	2.74	37.05	2.9
45	teja semha	450147d	60	43	58	96.66666667	53.666667	A	12.3	136	3.2	18	8.4	3.7	2.23		151	2.52	31.08	3.4
46	Suditi dwivedi	379943d	63	74	68	121	68.333333	A	8.2	135	6.2	21	6.7	6.5	9.9	226	1900	15.12	43.55	5.3

THESIS DATA SHEET

S no	name	id	lvids	lvpwd	rvdd	ivsd	fs	ef	e	a	lvmht	ABPM sbp	ABPM sbp1	ABPM sbp2	ABPM dbp	ABPM dbp1	ABPM dbp2	ABPM mbp	ABPM mbp1	ABPM mbp2
1	surya	813056b	3.9	1.2	0.89	0.98	20.9	42.4			1.4235151	122	123	117	80	80	77	92	92	89
2	meghana	028448f	2.7	0.68	1.2	0.91	34	63.4			0.89267385	113	114	107	71	72	68	86	87	84
3	alok yadav	81506d	2.2	0.7	0.73	0.73	31.3	60.4			0.53785167	116	119	108	67	72	54	85	88	75
4	amar kumar das	105944f	3.2	0.91	1.9	0.83	33.1	61.6			0.8557558	118	121	102	82	84	68	95	98	81
5	tanvir sheam	678614d	2.7	0.9	1	0.9	28.9	56.4			0.67835355	108	108	106	71	73	63	84	84	79
6	kiranjit biswas	785907d	2.9	0.84	0.92	0.95	32.7	61.5			0.892344	101	103	88	64	67	50	78	80	66
7	nazmus sakib	841588c	2.6	0.82	1.9	0.79	27.3	54	92.8	68.6	0.55086306	102	103	99	65	66	63	77	78	74
8	selv vinayagam	976429d	2.7	1	1.4	1.1	31.3	59.9			0.82809235	140	142	126	96	99	78	109	111	92
9	vignesh n	661380d	2.9	0.92	0.77	1.1	29.9	57.5			0.8718044	124	125	124	88	88	85	100	101	99
10	monisha	978513d	2.6	0.84	0.81	0.95	33.7	63.1			0.77046228	117	118	112	71	73	64	87	88	82
11	vishnu	310052d	2.6	1	0.98	1.5	29.6	57.5			1.42906569	104	105	99	68	69	65	80	81	75
12	shubham kumar	678186d	2.7	1.1	1	0.98	29.5	57.2	145	94.1	0.82195832	127	129	114	76	79	59	94	97	80
13	keshav ram sahu	030692f	4.1	1.1	1.3	1	12.8	27.5	150.5	71.1	1.50176656	123	123	122	89	88	95	100	100	104
14	premnath	979489b	2.5	0.98	1.1	0.87	39.1	70			0.84475723	135	135	139	86	86	86	99	100	99
15	prthibiraj barik	193478f	3.3	1.3	1.5	1.2	30	57.2			1.39837108	127	127	125	68	69	60	87	88	82
16	wahengbam dashibala	203196f	2.5	0.87	0.83	0.76	31.5	60.4	97.7	69.1	0.55263372	134	136	120	86	89	73	100	103	87
17	akhas dom	060141f	1.5	1.4	1.4	1.4	38	70.7			0.86149828	100	100	98	58	58	58	73	74	72
18	mohammed s	228209f	2.5	0.8	1	1	30.6	59	94.3	64.7	0.59026915	127	128	120	63	64	60	85	86	82
19	rozhan ali	047248c	2.4	0.7	1.1	0.8	31.4	60.4			0.63711111	96	96	98	58	58	55	71	72	69
20	raja v	539831d	2.7	0.61	0.65	0.45	31.7	60.4	101	71.8	0.46593369	137	137	138	86	86	86	104	104	104
21	rishi raghavendran	228705f	2.3	1	1.2	1	31.2	60.1	108	83.4	0.65859413	105	106	99	68	70	58	81	82	74
22	vijaya priya	608170d	2.3	0.79	0.98	0.87	32.8	62.3			0.51709082	110	111	105	72	73	65	85	86	78
23	anand s	134526c	2.5	0.92	0.77	0.65	30.7	59.3			0.57190274	116	116	110	76	77	71	89	90	83
24	bitan bera	239720f	3.4	1.1	1	0.9	20.9	42.9	50.8	49.9	1.0867884	134	137	124	96	98	86	108	109	98
25	parkavi	551548d	2.6	0.53	1.1	0.61	30.2	58.3			0.42304244	99	102	90	65	68	56	79	82	69
26	gangadhara	947648d	2.4	0.83	1.3	0.6	31.4	60.4			0.4777842	117	118	110	77	78	66	90	91	83
27	sudharson	896410d	3	0.77	1.4	0.92	29.8	57.2			0.72528918	118	120	109	74	76	64	89	90	81
28	debham saha	954703c	2	1.1	0.72	0.98	33.3	63.7	117	90.3	0.49372225	121	121	121	73	74	65	90	90	86
29	sudipta mondol	259172f	2.9		1.1	0.9	32.6	61.2	77.5	58.7	0.34672995	121	121	121	74	74	75	89	89	89
30	mohammed zaith	589689c	1.9	0.54	1.1	0.69	36.7	68.1	91.4	55.5	0.34443325	91	92	88	55	55	54	68	69	66
31	somojeet modak	260990f	2.9	0.5	0.5	0.5	31	59	90.3	75.5	0.41660179	92	94	83	60	61	51	72	73	62
32	roshan kumar	827840b	2.8	0.79	1.2	0.87	30.5	58.5			0.65833211	124	126	112	73	75	64	90	91	83
33	arvind	163511f	2	0.76	1.2	0.79	44.8	77	112.5	73.1	0.55371924	118	120	110	59	61	49	79	80	75
34	ravindran	152643c	2.7	0.76	1.3	0.61	31.3	59.9			0.56424737	108	110	98	62	63	59	78	79	74
35	furquanul haque	562142c	2.3	0.91	1.9	1.1	32.8	62.3			0.72617607	101	101	102	60	60	55	75	75	73
36	jatin sharma	220366f	2	0.6	0.9	0.8	35.5	66.4	97.7	57.8	0.48140082	110	112	99	79	81	71	90	91	82
37	soumitra manna	779138b	2.1	0.88	1.2	0.74	29.7	58.4	104.6	69.6	0.46940606	119	122	107	71	74	57	85	74	72
38	samuvel	045336c	2.3	0.7	1.2	0.8	34	64			0.52030935	106	106	102	71	72	62	83	84	79
39	Karthik	497918B	2.6	0.8	0.9	0.8	33.3	62.7	114	64.2	0.61336022	98	98	97	63	63	61	76	76	73
40	indumathy	852689d	1.9	0.79	1.1	0.89	32.7	63.1			0.51841402	91	94	83	54	56	48	68	70	61
41	niveda	132557d	2.4	0.7	0.9	0.6	40	71.2	91.8	54.8	0.51445488	110	111	103	69	70	66	83	84	77
42	adarsh	338748d	2.6	1	1.3	2.7	32.2	61.1	117	52.3	2.3892695	133	133	132	87	88	82	102	103	97
43	ramana	915292c	2.3	0.6	0.8	0.6	30.3	58.9	98.7	73.1	0.40204867	119	123	109	79	81	71	92	94	85
44	muniyappan	669607d	1.8	0.78	1.3	0.46	38	69.9	98.5	47.9	0.50942028	93	95	88	54	57	46	69	70	64
45	teja semha	450147d	2.3	0.72	0.68	0.91	32.1	61.4	96.7	59.2	0.70154597	95	96	91	57	58	51	69	70	65
46	Suditi dwivedi	379943d	3.7	1	1.1	1.1	30.5	57.5			1.56029711	133	134	128	86	86	85	99	100	98

THESIS DATA SHEET

S no	name	id	BP Load	LVH	LV Mass	NDS	NDD	SBP Index	DBP Index
1	surya	813056b	60.37	P	197.869	4.87805	3.75	1.0066	1.038
2	meghana	028448f	18.82	P	96.4088	6.14035	5.55556	0.97	0.958
3	alok yadav	81506d	57.69	P	58.6258	9.2437	25	0.996	0.9
4	amar kumar das	105944f	66.66	P	141.2	15.7025	19.0476	0.912	1.058
5	tanvir sheam	678614d	27.27	P	100.939	1.85185	13.6986	0.87	0.92
6	kiranjit biswas	785907d	7.27	P	122.251	14.5631	25.3731	0.841	0.832
7	nazmus sakib	841588c	10.81	A	79.3243	3.8835	4.54545	0.832	0.843
8	selv vinayagam	976429d	96.42	A	130.839	11.2676	21.2121	1.098	1.241
9	vignesh n	661380d	75	A	139.053	0.8	3.40909	0.973	1.138
10	monisha	978513d	40.74	A	104.398	5.08475	12.3288	0.994	0.958
11	vishnu	310052d	19.44	P	156.626	5.71429	5.7971	0.893	0.913
12	shubham kumar	678186d	53.57	A	129.047	11.6279	25.3165	1.01	0.984
13	keshav ram sahu	030692f	90.27	P	175.707	0.81301	-7.9545	1.053	1.16
14	premnath	979489b	84.74	P	123.081	-2.963	0	1.102	1.115
15	prthibiraj barik	193478f	34.48	P	232.13	1.5748	13.0435	0.982	0.877
16	wahengbam dashibala	203196f	89.28	A	80.6845	11.7647	17.9775	1.12	1.142
17	akhas dom	060141f	4.65	A	105.964	2	0	0.848	0.755
18	mohammed s	228209f	34.42	A	92.6723	6.25	6.25	1.01	0.816
19	rozhan ali	047248c	0	A	68.808	-2.0833	5.17241	0.77	0.766
20	raja v	539831d	93.33	A	52.6505	-0.7299	0	1.077	1.133
21	rishi raghavendran	228705f	10.63	A	98.7891	6.60377	17.1429	0.846	0.881
22	vijaya priya	608170d	24.63	A	79.1149	5.40541	10.9589	0.897	0.947
23	anand s	134526c	35.08	A	76.635	5.17241	7.79221	0.966	0.988
24	bitan bera	239720f	97.91	P	142.369	9.48905	12.2449	1.126	1.248
25	parkavi	551548d	4.34	A	52.6688	11.7647	17.6471	0.856	0.892
26	gangadhara	947648d	46.29	A	64.5009	6.77966	15.3846	0.975	1.001
27	sudharson	896410d	17.2	A	113.145	9.16667	15.7895	0.938	0.958
28	debham saha	954703c	14.1	A	82.9453	0	12.1622	0.922	0.946
29	sudipta mondol	259172f	13.58	A	51.316	0	-1.3514	0.975	0.959
30	mohammed zaith	589689c	0	A	40.9876	4.34783	1.81818	0.779	0.717
31	somojeet modak	260990f	0	A	55.8246	11.7021	16.3934	0.766	0.78
32	roshan kumar	827840b	28.57	A	98.0915	11.1111	14.6667	1	0.946
33	arvind	163511f	17.72	A	75.3058	8.33333	19.6721	0.983	0.767
34	ravindran	152643c	5.12	A	72.9008	10.9091	6.34921	0.908	0.806
35	furquanul haque	562142c	0	A	103.988	-0.9901	8.33333	0.824	0.778
36	jatin sharma	220366f	34.28	A	51.5099	11.6071	12.3457	0.925	1.051
37	soumitra manna	779138b	37.68	A	63.1821	12.2951	22.973	1.011	0.958
38	samuvel	045336c	17.14	A	65.663	3.77358	13.8889	0.899	0.924
39	Karthik	497918B	2.12	A	89.5506	1.02041	3.1746	0.8	0.817
40	indumathy	852689d	3.84	A	57.0255	11.7021	14.2857	0.781	0.728
41	niveda	132557d	24.07	A	71.0977	7.20721	5.71429	0.926	0.923
42	adarsh	338748d	75.75	P	305.826	0.75188	6.81818	1.118	1.131
43	ramana	915292c	68	A	46.3964	11.3821	12.3457	1.011	1.056
44	muniyappan	669607d	6.6	A	39.2254	7.36842	19.2982	0.798	0.725
45	teja semha	450147d	0	A	73.6623	5.20833	12.069	0.798	0.758
46	Suditi dwivedi	379943d	100	P	213.761	4.47761	1.16279	1.129	1.16